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CONTENTS OF VOLUME 9

JANUARY, 1923. NUMBER 1

PAGES

SECTION OF THE ANTEROLATERAL COLUMNS OF THE SPINAL CORD (CHORDOTOMY). DISCUSSION OF THE PHYSIOLOGIC EFFECTS AND CLINICAL RESULTS IN A SERIES OF EIGHT CASES. CHARLES H. FRAZIER, M.D., AND WILLIAM G. SPILLER, M.D., PHILADELPHIA.....	1
CLINICAL NOTES ON THE PATHOLOGY IN A CASE OF EPIDEMIC ENCEPHALITIS COMPLICATED BY A PSYCHOSIS. GEORGE B. HASSIN, M.D., CHICAGO, AND D. B. ROTMAN, M.D., DUNNING, ILL.....	22
NEUROSYPHILIS AMONG THE CHINESE WITH FINDINGS IN SIXTY-FIVE CASES. WILLIAM G. LENNOX, M.D., PEKING, CHINA.....	26
A STUDY OF FOUR CASES OF GLOSSOPHARYNGEAL NEURALGIA. JOHN B. DOYLE, M.D., ROCHESTER, MINN.....	34
LESIONS IN THE BRAIN OF A PATIENT WITH POSTENCEPHALITIC PARALYSIS AGITANS. J. CHARNLEY MCKINLEY, M.D., MINNEAPOLIS.....	47
THE PATHOLOGIC FINDINGS IN THE HEART MUSCLE IN PROGRESSIVE MUSCULAR DYSTROPHY. J. H. GLOBUS, M.D., NEW YORK.....	59
SEX DEVELOPMENT AND BEHAVIOR IN MALE PATIENTS WITH DEMENTIA PRAECOX. CHARLES E. GIBBS, M.D., WARD'S ISLAND, N. Y.....	73
SPIROCHETE STAIN IN MULTIPLE SCLEROSIS. GEORGE S. STEVENSON, M. D., WARD'S ISLAND, N. Y.....	88
ABSTRACTS FROM CURRENT LITERATURE	90
SOCIETY TRANSACTIONS	113
BOOK REVIEWS	132

FEBRUARY, 1923. NUMBER 2

GENESIS OF CEREBELLAR FUNCTIONS. FREDERICK TILNEY, M.D., NEW YORK	137
A CLINICOPATHOLOGIC STUDY OF ACUTE AND CHRONIC CHOREA. GEORGE WILSON, M.D., AND N. W. WINKELMAN, M.D., PHILADELPHIA.....	170
TWO CASES OF BRAIN TUMOR WITH VENTRICULOGRAPHY. PETER BASOE, M.D., AND CARL B. DAVIS, M.D., CHICAGO.....	178
THE DIFFERENCE BETWEEN A MUSCULAR AND NEUROMUSCULAR INTERPRETATION OF WALKING. WALTER M. KRAUS, M.D., NEW YORK.....	184
THE NATURE OF CERTAIN FUNCTIONAL NERVOUS DISTURBANCES AND THEIR TREATMENT ALONG METABOLIC LINES. RALPH PEMBERTON, M.D., PHILADELPHIA	208
ABSTRACTS FROM CURRENT LITERATURE.....	218
SOCIETY TRANSACTIONS	245
BOOK REVIEWS	281

MARCH, 1923 NUMBER 3

THE DEEP SENSIBILITY OF THE FACE. LOYAL E. DAVIS, M.D., CHICAGO...	283
AFFERENT IMPULSES OF THE TRIGEMINAL NERVE: THE INTRAMEDULLARY COURSE OF THE PAINFUL, THERMAL AND TACTILE IMPULSES. MARGARET W. GERARD, CHICAGO.....	306
HEREDITARY AND NONHEREDITARY MENTAL DEFECT: A COMPARATIVE STUDY. HOWARD W. POTTER, M.D., AND RUBY SMITH VIERS, M.D., LETCHWORTH VILLAGE, N. Y.....	339
A CASE OF AMBULATORY AUTOMATISM. H. DOUGLAS SINGER, M.D., M.R.C.P., CHICAGO	347
BASAL METABOLISM IN MENTAL DISEASE. K. M. BOWMAN, M.D., AND G. P. GRABFIELD, M.D., BOSTON	358
ABSTRACTS FROM CURRENT LITERATURE.....	362
SOCIETY TRANSACTIONS	388
BOOK REVIEWS	415

APRIL, 1923. NUMBER 4

TERATOID CYST OF THE HYPOPHYSIS. J. H. GLOBUS, M.D., NEW YORK.....	417
CHARCOT-MARIE ATROPHY: THE REPORT OF A CASE WITH NECROPSY. CHARLES S. POTTS, M.D., AND GEORGE WILSON, M.D., PHILADELPHIA...	431
RECENT DEVELOPMENTS IN ELECTRODIAGNOSIS. PERCIVAL BAILEY, M.D., PH.D., BOSTON	436

ARSPHENAMIN THERAPY IN STATE HOSPITALS FOR MENTAL DISORDERS. GEORGE W. MILLS, M.D., AND CHARLES L. VAUX, M.D., CENTRAL ISLIP, N. Y.	450
SCHIZOPHRENIC CATATONIA WITH ASSOCIATED METABOLIC AND VEGETATIVE FEATURES. THEOPHILE RAPHAEL, A. M., M.D.; JOHN PAUL PARSONS, B.S., M.D., AND MARGARET NEWELL WOODWELL, A.B., ANN ARBOR, MICH.	471
PHENOBARBITAL TREATMENT OF INSANE EPILEPTICS. IRA A. DARLING, M.D., WARREN, PA.	478
THE PATHOGENESIS OF EPILEPSY. MICHAEL OSNATO, M.D., NEW YORK....	488
OBITUARY: ROBERT THAXTER EDES, M.D.....	506
NEWS AND COMMENT	510
FORTY-NINTH ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION.—ANNUAL MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION.	
ABSTRACTS FROM CURRENT LITERATURE.....	511
SOCIETY TRANSACTIONS	527
BOOK REVIEWS	544

MAY, 1923. NUMBER 5

SOME PROBLEMS IN EPILEPSY. WALTER TIMME, M.D., NEW YORK.....	547
THE ECOLOGY OF EPILEPSY:	
I. EPILEPSY IN COLONIES. CHARLES L. DANA, M.D., NEW YORK.....	551
II. RACIAL AND GEOGRAPHIC DISTRIBUTION. C. B. DAVENPORT, PH.D., COLD SPRING HARBOR, N. Y.....	554
EPILEPSY AND THE CONVULSIVE STATE. FOSTER KENNEDY, M.D., NEW YORK	567
SYMPTOMATIC EPILEPSY IN BIRDS. G. MINGAZZINI, M.D., ROME, ITALY....	576
INFANTILE PROGRESSIVE MUSCULAR ATROPHY OF WERDNIC-HOFFMANN TYPE. JOSEPH C. MICHAEL, M.D., MINNEAPOLIS.....	582
CYSTIC BLASTOMYCOSIS OF THE CEREBRAL GRAY MATTER CAUSED BY TORULA HYSTOLYTICA STODDARD AND CUTLER. WALTER FREEMAN, M.D., AND F. D. WEIDMAN, M.D., PHILADELPHIA.....	589
EXPERIMENTAL CONVULSIONS. LEWIS J. POLLOCK, CHICAGO.....	604
STUDIES IN EPILEPSY. CONVULSIONS EXPERIMENTALLY PRODUCED IN ANIMALS COMPARED WITH CONVULSIVE STATES IN MAN. CHARLES A. ELSBURG, M.D., AND BYRON P. STOOKEY, M.D., NEW YORK.....	613
METABOLIC STATES CONTRIBUTING TO UREMIA. NELLIS B. FOSTER, M.D., NEW YORK	627
NEWS AND COMMENT:	
THE STATUS OF EPIDEMIC ENCEPHALITIS.....	633
THE TEST OF HYPERTHYROIDISM.....	635
A CORRECTION	636
ANNUAL MEETINGS	636
ABSTRACTS FROM CURRENT LITERATURE.....	637
SOCIETY TRANSACTIONS	652
BOOK REVIEWS	673

JUNE, 1923. NUMBER 6

THE MECHANISM AND THE FUNDAMENTAL CAUSE OF THE EPILEPSIES. JOSHUA ROSETT, M.D., NEW YORK.....	689
LESIONS OF THE OPTIC CHIASM AND TRACTS WITH RELATION TO THE ADJACENT VASCULAR STRUCTURES. TEMPLE FAY, M.D., AND FRANCIS C. GRANT, M.D., PHILADELPHIA.....	739
EPIDEMIC ENCEPHALITIS. A PATHOLOGIC STUDY OF FIVE CASES INCLUDING TWO WITH MYOCLONIA. M. E. MORSE, M.D., BOSTON.....	751
STABILIZING BRAIN TISSUES DURING FIXATION. GEORGE S. STEVENSON, M.D., VINELAND, N. J.	763
IMPLANTATION OF BACILLUS ACIDOPHILUS IN PERSONS WITH PSYCHOSES. L. A. JULIANELLE, M.D., AND F. G. EBAUGH, M.D., PHILADELPHIA....	769
OBITUARY: EDWARD MERCUR WILLIAMS, M.D.....	778
NEWS AND COMMENT:	
THE THERAPY OF EXOPHTHALMIC GOITER.....	781
ABSTRACTS FROM CURRENT LITERATURE.....	783
SOCIETY TRANSACTIONS	803
INDEX TO VOLUME.....	831

Archives of Neurology and Psychiatry

Vol. 9

JANUARY, 1923

No. 1

SECTION OF THE ANTEROLATERAL COLUMNS OF THE SPINAL CORD (CHORDOTOMY)

DISCUSSION OF THE PHYSIOLOGIC EFFECTS AND CLINICAL
RESULTS IN A SERIES OF EIGHT CASES *

CHARLES H. FRAZIER, M.D., AND WILLIAM G. SPILLER, M.D.
PHILADELPHIA

There are few conditions the physician has to treat which cause so much distress as that of intense pain uncontrolled by drugs. A means of relief in selected cases is afforded by chordotomy. The operation will not be entirely successful in every case in which it is employed—some pain fibers occasionally will escape the knife—but the operation has now been performed sufficiently often to justify a belief in its efficacy. It is a delicate procedure, requires a skilful technic, and is not to be undertaken lightly by one untrained in the surgery of the spinal cord. It requires on the part of the operator a visualization of the microscopic anatomy of the cord, and a realization that a slight misplacement of the incision may cause motor paralysis of one or both lower limbs, depending on whether the division is unilateral or bilateral. We¹ have written on this subject previously and therefore do not refer to the literature at the present time.

We report in this paper eight cases in which chordotomy has been performed. All but one have not been reported previously, but in this one case chordotomy for certain reasons mentioned in the notes has been performed three separate times on the same side of the cord. In one case (Case 8), the operation was performed to remove the intense hyperesthesia above the anesthetic area produced by transverse myelitis, and was entirely successful.

The operation abolished the pain for which it was performed in six of these cases, and gave decided relief from pain in the remaining

* Read at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

1. Spiller, William G., and Martin, Edward: The Treatment of Persistent Pain of Organic Origin in the Lower Part of the Body by Division of the Anterolateral Column of the Spinal Cord, *J. A. M. A.* **58**:1489 (May 18) 1912. Frazier, Charles H.: Surgery of the Spine and Spinal Cord, New York, D. Appleton & Co., 1918, chapter 6. *Ibid.*: Section of the Anterolateral Columns of the Spinal Cord for the Relief of Pain: Report of Six Cases, *Arch. Neurol. & Psychiat.* **4**:137 (Aug.) 1920.

two cases. Four of these were cases of malignant tumor. In one of these, in which the suffering had been excruciating and constant, the relief was so intense that the patient expressed herself by saying she felt as though she were in heaven. In one case the childishness of the patient interfered with careful study, but there was reason to believe that much relief from suffering had been obtained. One case occurred in an army officer who had been injured in the war. Chordotomy was performed three times over a period of nearly three years. After the first operation relief from pain was great for a period of about three months. Whether the relief from this first operation was felt for a longer time than after the later incisions of the cord because the operation then was new to the patient, cannot be asserted, but this is possible. More than two years later the chordotomy was repeated with relief again, but not with complete cessation of pain. It was then found that certain fibers innervating a small strip on the inner side of the leg below the knee and on the inner sole of the foot, that is, in the distribution of the long saphenous nerve, must have escaped in the section of the cord, as it was possible to produce pain here by pressure, but the patient could not localize the pain so produced to the part irritated.

The long saphenous nerve was cut to determine whether the pain was of peripheral origin and could be relieved without further operation on the spinal cord. For a short time this procedure relieved the pain. A third chordotomy was performed lower in the cord, at the tenth thoracic segment, in the hope that if any fibers had escaped in the two previous chordotomies, they might occupy a different situation lower in the cord and be caught by the section of the anterolateral column here. The persistence of pain in a modified form in this case was so unlike the results in the other cases that we were perplexed. Certain observations convinced us that a considerable psychic element was present. It was found that sugar given at certain times to relieve pain did so promptly when the patient believed he was obtaining more powerful remedies.

We had no reason to believe that hemorrhage occurred into the spinal cord after chordotomy in any case, except possibly in one, in which some transitory paresthesia occurred in the foot three or four days after a second chordotomy and some transitory dysesthesia and blunting of temperature sensation but not of pain sensation in the lower limb after the third chordotomy, on the side of the operation. Whether this sensory disturbance on the side of the operation was caused by hemorrhage or edema or by some other lesion, we have no means of determining, but is probable that slight transitory impairment of function was caused in the side of the cord opposite to the chordotomy at the time of the operation by the necessary manipulation.

We feel convinced that no pain or temperature fibers, however, ascend in the spinal cord uncrossed, as in two other cases in which unilateral chordotomy was performed no disturbances for these forms of sensation developed in the lower limb on the side of the operation. On this side, pain, heat and cold sensations were normal after unilateral chordotomy. We feel convinced that this operation, either unilateral or bilateral, does not distinctly impair tactile sensation in the lower limbs, and therefore our observations confirm the opinion that for impairment of tactile sensation the lesion must destroy more than the anterolateral columns.

We are convinced that this operation when successfully performed destroys pain, heat and cold sensations in the lower limbs and lower part of the trunk when performed at the proper level, although the impairment of these sensations is less intense over the abdomen in some cases, probably because some fibers have not decussated at the level of the incision. In some cases we have observed that certain fibers innervating the lower limbs for pain and temperature sensations escape in the division of the anterolateral columns. This is not surprising, because in the endeavor to spare the crossed pyramidal tract it is possible that a few sensory fibers for pain and temperature escape.

We are impressed by the fact that the bladder and rectum almost invariably escape damage in this operation. In only one of these cases (Case 3) in which a more liberal section of the cord was made deliberately, was there loss of bladder control while the patient was in the hospital; in this case, too, partial paralysis of the lower limbs occurred, probably as a result of injury of the crossed pyramidal tracts. One might possibly expect sympathetic fibers for the bladder and rectum to be in the anterolateral columns and to be impaired by this operation, but it has seemed to us that when such disturbance occurs from a spinal cord lesion the crossed pyramidal tracts are damaged. We are inclined to believe that in the male impairment of sexual power possibly may result in the manner suggested. Our basis for this hypothesis is that in one case in which unilateral chordotomy was performed sexual power was lost. We mention this, feeling that one case is not sufficient to determine this matter.

The marked impairment of pain and temperature sensations after chordotomy at the fifth thoracic segment usually is pronounced to about 2 inches (5.08 cm.) above a line passing through the umbilicus, and gradually shades off above this region. We do not believe that pain sensation is conveyed in the spinal cord in association with deep pressure sensation. While pressure sensation is preserved, it is without any association with pain sensation.

Clinical observations have shown us that disease in the spinal cord is able to produce a dissociation of pain and temperature sensations,

but our observations have shown that division of the anterolateral columns produces, when successfully performed, a loss of pain, heat and cold sensations, from which we must conclude that fibers conveying these sensations are closely associated in the anterolateral column. We have observed, for example, in a case of extramedullary tumor of the midcervical region, that heat and cold sensations were greatly impaired on the opposite side of the body, while pain sensation was normal in this part. After chordotomy we have noticed that occasionally temperature sensation is lost and pain sensation greatly impaired.

Chordotomy seems peculiarly adapted to cases of malignant tumor and affords relief to the intense suffering from this affliction. It is no small satisfaction to bring comfort to one afflicted with an incurable tumor who is conscious of approaching death and is grateful for any means of making life endurable.

It is striking that there has usually been no serious implication of the motor fibers to the lower limbs. In one case (Case 3), in which the incision was more extensive because of the necessity of relieving unbearable pain, there was partial paralysis of the lower limbs. The possibility of this form of paralysis must always be borne in mind.

We have felt greatly gratified that we have had no reason to believe that extensive hemorrhage or edema of the spinal cord has resulted from the operation, although we have mentioned that in one case slight postoperative hemorrhage into the cord substance was possible. In no case was there visible hemorrhage at the site of section.

We are not surprised at the occurrence of some pain in the trunk at the level of the operation following the chordotomy and lasting a few days to a week or even longer, and on the side of the division of the anterolateral column, or even on the opposite side. This pain probably is caused by the manipulation of the spinal cord in the exposure of the anterolateral columns; it is important, therefore, that the spinal cord should be disturbed as little as possible during the operation.

We have found unilateral chordotomy sufficient for pain strictly confined to one side of the body. It is not necessary to perform bilateral chordotomy in such a case, but when the lesion is likely to implicate the opposite side of the body speedily, bilateral chordotomy is desirable, even though the pain may be unilateral at the time of operation. Some preservation of objective pain sensation does not imply necessarily that the spontaneous pain has not been removed, but it is always more satisfactory to find a complete loss of objective pain sensation in the previously painful part after chordotomy.

We have not observed trophic disturbance other than the existence of bedsores in only two of the cases, and in one of these (Case 3), microscopic examination of the spinal cord after death showed that the

crossed pyramidal tracts were seriously implicated. While there were no symptoms of pyramidal tract lesion in the other case, we believe that some injury of this tract may have resulted, even though the symptoms did not indicate this. At all events, we do not believe bed-sores are likely to develop from division of the cord confined to the anterolateral region.

REPORT OF CASES

CASE 1.—History.—J. F. C., aged 33, referred to the neurosurgical service, University Hospital, by Dr. Foster Kennedy, Feb. 9, 1922, in September, 1916, was struck in the third and fourth lumbar vertebrae by a high explosive missile and completely paralyzed from the waist down, including the sphincters. About thirty-six hours later the missile was removed. Great pain down the legs persisted. One year before admission two operations were performed on the patient elsewhere. At these operations, it was reported a number of posterior roots on the left side of the cord were cut. Following these operations, there was loss of sensation over a considerable area of the lower extremities, but the pain continued as before. Pain was always present, chiefly in the left knee and right ankle, but there were exacerbations of intense severity. It was shooting in character and at times agonizingly severe. The pain in the right ankle could be controlled by morphin, but not the pain in the left knee. At times it was even more severe in the left groin between the area of normal and of reduced sensation. Pressure and especially pin prick in this area started paroxysms of intense pain shooting down the inner side of the thigh on the left side. Occasionally it affected the left testicle. The patient had become tolerant to morphin, taking from 4 to 8 grains (0.26 to 0.52 gm.) a day. He had stopped taking it at various times during the last five years, once for a year and at other times for several months, but he had taken it continuously for nearly a year before admission. Mental vigor was unimpaired. Sexual power was present until the most recent operation, when sensation was abolished from the anterior half of the penis.

Examination.—The patient was a well developed, well nourished man. He was restless, sometimes talkative, sometimes apprehensive, and at frequent intervals stopped any occupation and cringed under excessive pain in the legs, the face becoming slightly flushed and the pulse slow. When the paroxysm had passed he talked again in a controlled voice. Extremities: The upper extremities were normal. There was atrophy and flaccid paralysis in all muscle groups in the lower extremities. There was slight power in the flexors and adductors of the right thigh, but none in the leg. The feet were in the equinus position. Patellar and Achilles' reflexes were lost. Tapping the adductor tendon produced distinct contraction on the right but not on the left.

Objective Sensation: Below the iliac crest on the left, and extending down from the buttock on the right and including the foot there was anesthesia to light touch and to pain, except for a small area over the internal malleolus which was hypersensitive. A narrow band on the right and a broad one on the left above the level of anesthesia, were hypersensitive to light touch, and especially to pin prick. On the left side, pin prick here initiated a severe spasm of pain down the left leg.

The genitalia and anus were insensitive.

Subjective Sensation: The most intense pain was felt in the left knee and the right foot along its outer edge and in the abdomen at the junction of the anesthetic and the normal sensory area especially on the left side, but in the latter place there was never pain unless the patient was examined or stimulated over this part. The inner side of the right ankle was sensitive to touch or pin prick.

Psychic effects were manifested by pain in the lower limbs. Any emotional disturbance, such as anxiety or great sympathy, caused pain in the lower limbs.

Pain occurred in either lower limb, but never in both at the same time. The pains began in the feet and surged up, on the left side, into the testicle. Pain was felt to the same level on the right but did not involve the testicle.

Bladder: By recording the amount of fluid that he had taken and the time which had elapsed, the patient knew when the bladder should be emptied, and by voluntary contraction of the abdominal muscles he could completely evacuate the bladder, especially when lying on his right side. The abdominal muscles were greatly hypertrophied and unusually strong. He had no movement of the bowels without drugs.

Operation and Course.—On Feb. 20, 1922, laminectomy and section of the anterolateral columns of the cord were performed, at the level of the fifth thoracic segment on the right side and at the level of the sixth segment on the left.

Feb. 26, 1922: The patient had little pain; the pain in his right lower limb was entirely gone, as well as most of the pain in the left lower limb.

Beginning two weeks after the operation the narcotics were gradually withdrawn without the patient's knowledge, and during the last two weeks the patient was in the hospital he was receiving no opiates.

March 16, 1922 (Dr. Spiller): Pin prick over the right thigh usually produced a sensation of pressure and occasionally of pain, but the pain sense was not normal and was delayed (that is, felt after the pressure). The same was true of the right side of the abdomen as high as the umbilicus. In the left lower limb pain sense was entirely lost and pain sensation was lost in the left side of the abdomen to about 2 inches (5.08 cm.) below the umbilicus.

Heat and cold sensations were lost over the right lower limb and the right side of the abdomen to about 1 inch (2.54 cm.) above the umbilicus, and were lost in the left side of the abdomen to about 2 inches below the umbilicus. The pain sensation was a trifle more acute over the right lower part of the abdomen than over the right thigh.

There was frequent involuntary jerking of the right lower limb, independent of any purposive irritation.

March 30, 1922: The patient was discharged, having been completely relieved of pain. All drugs were withdrawn.

April 11, 1922: A letter was received from the patient in which he said: "I really can't thank you for relieving me of so much pain, but I must make the attempt. The pain has left me in a marvellous degree."

CASE 2.—History.—G. V., aged 50, admitted to the neurosurgical service, university Hospital, June 28, 1921, referred by Dr. G. M. Piersol, presented symptoms of a spinal cord tumor at the level of the ninth thoracic segment. November 22, 1921, an exploratory laminectomy revealed a sarcoma of the vertebral column involving the spinal canal and pressing on the cord. That portion of the tumor within the canal was removed. Because of the persistent and agonizing pain, a chordotomy was advised.

Operation and Course.—Dec. 10, 1921, section of anterolateral columns was performed at the level of the fifth thoracic segment. The spinous processes and laminae designed to uncover the fifth thoracic segment were removed. The anterolateral columns on both sides were sectioned by our customary technic, that on the right side about 1 cm. higher than the left. In other particulars the operation was carried out without modification of the usual technic. A little oozing from the artery which accompanies the posterior root on the left side presented the only difficulty.

Dec. 20, 1921 (Dr. Spiller): Heat and cold were not recognized on the right side of the abdomen for 10.5 cm. above the upper margin of the umbilicus, and on the left side 7.5 cm. above the upper margin of the umbilicus. Pain sensation was lost on the right side of the abdomen to 2 cm. below the lower margin of the umbilicus; occasionally pin prick was recognized on the right side below this line in an indefinite manner. Above this line on the right side pin prick was felt, but apparently not in a normal manner until the level of the thermo-

anesthesia was approached. On the left side pin prick was not felt as high as the area of thermo-anesthesia, although occasionally it was recognized over the lower part of the left side of the abdomen in an abnormal manner. The patient was decidedly childish in her answers and tests had to be made with great care.

From the time of the operation until the patient's discharge, she was entirely relieved of the pain of which she complained before the operation. Before the operation the slightest jar of the bed caused excruciating pain; after the operation, none at all. The patient suffered somewhat from root pains at the level of the chordotomy. These have been observed after other similar operations but are only of a transitory nature.

CASE 3.—*History*.—F. S., aged 49, referred to the neurosurgical service, University Hospital, by Dr. D. D. Reynolds, Dec. 19, 1921, had her right breast amputated, April, 1919, and a resection of the left breast. The growth in the left breast recurred and she received a course of roentgen-ray treatment. In April, 1921, she began to complain of pain in the calf of the right leg and later from the knee to the ankle, chiefly on the inner side. Later the pain spread to the outer side of the leg, to a narrow zone in the back of the thigh and to the foot. It felt as though something were pressing on the sole of the foot. Later she experienced pain in the sacral region and across the buttock. November, 1921, pain began in the left lower extremity, particularly on the inner side and front of the thigh, but also in the back of the thigh, down below the knee and occasionally in the foot. She had severe pains across the lower part of the back at times and in the rectum and vagina. The pains were sharp and shooting and almost constant, although there were occasional remissions. Recently the patient suffered from pain of a neuralgic character over the left eye. She had lost little weight and felt that were the pain gone she would be well and able to take care of her house.

Dec. 23, 1921: Both patellar reflexes were very prompt, but equal and a little exaggerated. No Achilles' reflex was obtained on the right side. A normal Achilles' reflex was present on the left. Laségue's sign was distinct on the right; it was slight on the left. Pain was present in both hip joints, not so severe in the left, and it followed the sciatic nerve on the right; but there was more in the front of the thigh than on the left. It was painful to sit on the right buttock. No bladder or bowel disturbance was present. She was never without a sensation of discomfort, and pain was severe every day, especially toward evening. Moderate degrees of heat and cold were promptly and correctly recognized in all parts of the lower limbs and over the whole of the abdomen and chest. The point was always recognized correctly from the head of the pin in the same parts. At times she did not seem to have acute pain sensation over the abdomen, but there was probably no real loss of pain sensation.

Roentgen-Ray Report: There was a metastatic carcinoma of the fifth lumbar vertebra and both iliac bones, especially on the right side, and probably of the upper part of the right femur. Chest: There was a mediastinal shadow which was suspicious; the right diaphragm was very high, suggesting metastatic growth in the liver.

Dec. 21, 1922: Bilateral section of the anterolateral columns was performed. Part of the first, all of the second, third, and part of the fourth thoracic spinous processes and laminae were removed. This gave ample exposure to the fourth and fifth thoracic segments. We were a little uncertain at the time as to whether we had exposed the fourth and fifth or the fifth and sixth thoracic segments, as two pairs of roots seemed to come off opposite the base of the third thoracic vertebra, one opposite the upper portion and the other opposite the lower portion, and it was at these respective levels at a distance of 1 and 0.7 cm. apart, that the sections were made, the higher section on the right, the lower section on the left. Not a drop of blood escaped within the spinal canal during the operation. Section was made as far as

could be determined by observation, 3 mm. deep and broad, the knife penetrating at a point midway between the posterior and anterior roots and coming out at the anterior roots.

Dec. 23, 1921: Heat and cold sensations began to be clearly recognized on the abdomen about 1 inch above the upper level of the umbilicus on the right side. The area on the left side was not determined because the patient was too tired. There was no clear perception of temperature sensation in either lower limb. Occasionally, she answered correctly for temperature over the lower part of the abdomen on either side. It is uncertain whether this was a guess or not. Pin prick caused no pain below the knee on either side. On the front and inner side of each thigh she recognized pin prick occasionally, but it did not seem to produce the usual pain. She rotated the left lower limb at the hip, had slight flexion of the toes of her left foot but no extension of the toes and no power at the ankle. The same was true of the right lower limb, but the voluntary power was not quite as great in the left lower limb.

Dec. 31, 1921 (Dr. Spiller): There was no distinct recognition of pin prick over the front and inner side of either thigh, although she answered correctly occasionally concerning pin prick of these parts. Pin prick was felt indistinctly over the lower part of the abdomen on either side, but it required a fraction of a minute before she was able to form an opinion of the irritation, and the pin prick was not as sharp as it was at higher levels where it was recognized instantly. This hypalgesic area extended to about $1\frac{1}{2}$ inches (3.81 cm.) above the upper level of the umbilicus on the right side and a little higher on the left side. It shaded off gradually into the normal area. She had not had any pain in her lower limbs since the operation. She said that before the operation she "suffered tortures," and that her condition after the operation was "like heaven," and that it was "marvelous." She had had three months of terrible suffering, waking on account of the pain after dozing a short time, even after morphin. She was most enthusiastic in her expressions about the relief of pain.

Since the operation she had had incontinence of feces. For nine days she had had retention of urine and was catheterized every twelfth hour. On December 31 she had incontinence of urine.

Jan. 5, 1922: Power in the lower limbs was much greater than immediately after the operation.

Jan. 18, 1922 (Dr. Spiller): She was able to flex the leg on the thigh and draw the foot up on both sides. This was done with effort. There was difficulty in dorsiflexion of the foot and toes.

Tactile sensation was normal everywhere about the lower extremities.

Heat and cold sensations were not entirely lost anywhere in the lower limbs, but were greatly disturbed in the lower limbs and over the abdomen. Extremes of temperature were occasionally correctly recognized in the lower limbs and still better over the abdomen.

There was great disturbance of temperature sensation to a level about 3 cm. above the umbilicus on the right side and somewhat higher on the left side. There was not a sharp line of definition.

Pain sensation was affected in about the same manner as temperature sensation. A deep pin prick was occasionally recognized as sharp over the lower limbs and still more so over the abdomen to about the same height as the disturbed temperature sensation on the right side and about 7 cm. above the umbilicus on the left.

The shading off of the area of anesthesia for pain and temperature was so gradual over the abdomen that it was impossible to make any sharp definition.

She could draw up either lower limb at the hip or knee and extend it. She could also flex the toes of each foot, but had no power in dorsiflexion of the ankle or the toes.

The patellar reflex was very feeble; indeed it apparently was absent on both sides even with reinforcement. The Achilles reflex was absent on both sides. The lower limbs were distinctly flaccid.

She still had a little pain between the shoulders in the region of the operation, but since the operation she had had no pain in the lower limbs or in the abdomen. She had had a good deal of distention of the abdomen, and when this had occurred she had had a great deal of discomfort. This was not like the pain from which she had suffered previously.

She was most enthusiastic over the relief from the intense pain she had experienced before operation.

Jan. 19, 1922: The patient was discharged.

A letter received from the patient's husband, dated March 20, 1922, stated that the operation had caused complete cessation of the pain in the lower limbs. The operation, therefore, was entirely successful in the removal of the pain for which it was performed. The patient had to be catheterized.

Necropsy Examination.—Removal of the spinal cord by Dr. Grant was permitted after death. Carcinoma nodules were found on the roots of the cauda equina. A transverse section of the cord at the fourth thoracic level, stained by the Marchi method, showed considerable degeneration of the peripheral portion of the anterolateral columns and of the anterior portion of the direct cerebellar tracts. The portion of the white matter near the anterior horns did not show recent degeneration, and evidently these fibers do not degenerate upward. The section was above the area of operation. These fibers were degenerated below this level. A section made at the sixth thoracic level showed considerable degeneration of the crossed pyramidal tracts and of the white matter adjoining the anterior horns, even in the anterior columns, but not much of the periphery of the vertebral columns.

CASE 4.—History.—Mrs. W. W. K., aged 34, referred to the neurosurgical service of the University Hospital by Dr. A. M. Ornsteen and Dr. William G. Spiller, Nov. 19, 1921, was well until five years before, when she began to void at frequent intervals, with pain during micturition. About one and one-half years after the onset she began to have pain in the left side of the vagina and rectum, also in the sole of her left foot. These pains were shooting in character, and came on at intervals of a few days and lasted for several days. During some of these attacks, she would have to stay in bed, and walking would make the pain worse. The condition became worse, and in the summer of 1920 she was operated on by Dr. Floyd Keene, who removed two Hunner's ulcers from the bladder. She had been slightly better since. She voided every hour during the day and night and had considerable pain doing so. Lying on her back gave her pain in the left sacral region. The pain was controlled only by morphin. She took almost 2 grains (0.13 gm.) a day.

Examination.—Deep reflexes in the upper extremities were normal. Patellar reflexes were exaggerated and equal. Ankle reflexes were exaggerated, the left more so than the right. Sensation was practically normal, except for a slight hypesthesia and hypalgesia in the fifth sacral segment (anal region).

Operation and Course.—Nov. 26, 1921: Section of the anterolateral column at the level of the fifth thoracic segment was performed.

The spinous processes and laminae of the fourth and fifth thoracic vertebrae were removed so as to expose the fifth thoracic segment. When the dura was opened, that portion of the cord, including the right anterolateral column, was grasped with the needle and section made with a knife, 2.5 mm. deep. The knife was plunged straight in so that the section was square. At first it appeared as though the section might have been too deep, at least the diameter of the cord at this level was quite small and the needle looked relatively large.

Nov. 29, 1921 (Dr. Spiller): The patient had pain in the right side of the vagina and rectum for twenty-four hours after the operation. On November 29, she complained of pain in the lower part of the right side of the abdomen. She had pain over the right side, partly as a result of the operation. The right lower limb was a little paretic. The patellar reflex on the right side was a little prompter than normal; she had right abortive ankle clonus and a typical Babinski reflex, but moved the entire right lower limb freely, even the toes.

For twenty-four hours after the operation she did not move the right lower limb. She had complete loss of heat and cold sensations over the whole left lower limb and abdomen up to 5 inches (12.7 cm.) above the umbilicus and extending close, but not fully to, the median line. Touch sensation was normal on the left side.

Dec. 1, 1921 (Dr. Spiller): The loss of pain, heat and cold sensation was complete on the left side to about 2 inches (5.08 cm.) above the umbilicus. The pain on the left side had stopped completely, but she complained much of pain over the right side of the trunk extending into the right side of the vagina; the pain was worse in the lower part of the right side of the abdomen. This pain may have been caused by the operation and referred from the site of operation, or it may have been caused by lying on the abdomen during the operation. The impairment of motion in the right lower limb had almost disappeared; the right knee reflex was a little exaggerated, but there was no ankle clonus on the right, although there was a typical right Babinski reflex. She moved the entire right lower limb and toes freely. The loss of pain and temperature sensations on the left side of the abdomen was well defined. She had no diminution of pain and temperature sensations in the right lower limb or abdomen.

Dec. 3, 1921 (Dr. Spiller): All pain in the lower part of the right side of the abdomen stopped December 2, and on December 3 she had full control over the right lower limb.

Dec. 10, 1921 (Dr. Spiller): The patient left the hospital on this date. She limped a little on the right, but the old pain had not been present since the operation. Movement in the lower limbs was normal, except that she dragged the right lower limb a little as she walked. There was no change in the reflexes.

March 4, 1922: The pain for which the operation was performed had not returned.

Sept. 14, 1922: Pain had persisted in the right side of the vagina, right side of the bladder, outer side of the right thumb, and heel and posterior half of the outer border of the right foot, and chordotomy of the left anterolateral was necessary. The operation had removed all pain from the left side of the body, and it was performed to accomplish this. She had a little subjective weakness of the right lower limb, felt only when she tried to hurry in walking.

CASE 5.—*History*.—Mrs. H. W., aged 37, referred to the neurosurgical service of the University Hospital by Dr. Curtis Burnam and Dr. R. M. Lewis, April 22, 1921, in August, 1917, had had a colostomy performed for rectal carcinoma. December, 1917, radium therapy had been administered under the direction of Dr. Howard Kelly. In January, 1918, a modified Kraske operation had been performed by Dr. Kelly. There was general improvement until May, 1919. From that time to April, 1920, her condition became worse, and she began to use opiates. Since then the pain had become more and more unbearable, and she had resorted to increasing doses of opiates. The pain was referred to the sacrum and down the right leg, sometimes to both legs. The pain exceeded her endurance and she found life unbearable. Before operation she required 3 grains (0.19 gm.) of morphin daily.

Examination.—The patient was emaciated and appeared cachectic. The lower extremities were markedly atrophied, and the patellar reflexes exaggerated.

Operation and Course.—April 30, 1921: Section of the anterolateral columns, right and left, at the level of the tenth thoracic segment was performed. The spinous processes of the ninth, tenth and eleventh and the laminae of the tenth and eleventh thoracic segments were removed. The cord was rotated first in one direction, then in the other, and with the special needles the anterolateral columns were isolated and sectioned.

The day after the operation the patient complained of a sensation of constriction as by a tight band about her waist at the lower margin of the ribs. At times for several days she had a sense of suffocation, which was difficult to explain.

The wound healed by first intention, and the stitches were removed on the sixth day. On the eighth day, the patient was up in a wheel chair. Ten days after the operation all morphin had been withdrawn, and the patient was free from pain. She could not appreciate the fact that her pain had been entirely subdued.

Eleven days after operation, the patient became drowsy, and on the thirteenth day she died of meningitis. The relief from pain in this case was striking. The development of a delayed infection almost two weeks after the operation was hard to explain, except on the grounds that in the cancerous cachexia of four years' duration the patient's resistance had been lowered. Subsequently, she was free from pain, but no objective examination had been made as to loss of pain and temperature sense.

CASE 6.—History.—A. B., aged 53 years, was admitted to the neurosurgical clinic of the University Hospital, Oct. 28, 1921. In August, 1919, a carcinoma of the cecum was removed. There were no adhesions and apparently no enlarged glands. The patient made a good recovery, and was well until February, 1921, when she began to have pain and paresthesia in the distribution of the right external cutaneous nerve—much like meralgia paraesthetica—and soon anesthesia and analgesia. A second exploration in April, 1921, revealed an inoperable mass.

The pain was constant most of the time, severe, aggravated by walking, and was wearing out the patient. Morphin was not tolerated; it caused nausea and vomiting. If the patient were free from pain, life might be prolonged a considerable time in comparative comfort, as there was no evidence of intestinal obstruction. With this understanding and with this in mind, the patient was referred by her physician to Dr. Frazier for a chordotomy.

Operation and Course.—Oct. 31, 1921: A chordotomy of the left anterolateral column was performed. The laminae and spinous processes of the fourth and fifth thoracic vertebrae were removed. By traction on the dentate ligament, the cord was slightly rotated. The special curved needle was introduced at a point midway between the posterior and anterior roots, and made its exit at the anterior root. The cord tissue included in the curve of the needle, representing a section 2.5 mm. broad and 2.5 mm. deep, was divided with a cataract knife. There was no bleeding.

Nov. 12, 1921: Tactile sensation was normal in the right lower extremity, except in the former area of anesthesia. Pain sensation was not felt distinctly as pain, but was felt sufficiently to permit recognition of the point from the head of the pin. This sensation was at times one of pricking from the point of the pin or of scratching by the fingernail, but was less acute than that of a pin prick on the other limb; it was readily exhausted. While fairly acute at the beginning, the recognition of the pin point was lost for most pricks made later. This disturbance of pain sense was present in the whole right lower limb and also, though less marked, over the right side of the abdomen about up to the level of the umbilicus. Over the right side of the abdomen pain sensation was less affected than in the right lower limb and also less affected about the level of the umbilicus. In this right abdominal area, pin prick was usually recognized, but much less acutely than on the left side. On the left side the plantar reflex was normal; ankle and knee reflexes on the left were a trifle more prompt than normal; possibly there was an abortive ankle clonus on the left. Thermal: Ice and hot water were not recognized on the entire right lower extremity. All over the lower part of the right side of the abdomen ice was called pin prick to 2 or 3 inches above the umbilicus, and very hot water was occasionally recognized correctly, but with much less intensity than over the corresponding area on the left side.

Dec. 12, 1921: Pain, heat and cold sensations were lost in the right lower limb, immediately recognized on the right side of the abdomen above Poupart's ligament, but not so keenly, and these sensations became more acute as the region of the umbilicus was approached. On the right side of the abdomen

she confused heat and cold sensations. She could not distinguish one from the other clearly to about 3 inches above the umbilicus. While she recognized pin prick on the right side of the abdomen, the sensation was not as sharp as on the left side of the abdomen. The impairment of temperature sensation extended a little higher than impairment of pain sensation on the right side of the abdomen. There was no weakness of the left lower limb, and no upward movement of the left big toe in the Babinski reflex. There were no diminution of pain, heat and cold sensations in the left lower limb. She had "creeping sensation," that is, some numbness from the right hip to the knee.

She had had almost entire relief from pain on the right side since the operation.

She had had a sense of fatigue in the left side of the abdomen and the left hip since the operation; this was not a true pain, but more in the nature of an ache, and was sometimes relieved by changing the position.

Feb. 19, 1922: There was an indefinite area about the inner and anterior aspect of the knee, in which a needle prick was distinctly perceived as sharp, and was decidedly uncomfortable. The analgesia extended to about the inguinal region, but the horizontal upper border was not sharp. Above that, as high as the level of the umbilicus, there was an area in which pain sense seemed rather less acute on the right than on the left, but testing with a sharp needle showed there that pricks were painful. There had not been a return of spontaneous pain in the right lower limb. Indeed, there had not been severe pain anywhere.

CASE 7.—*History*.—H. E. H. D., aged 47, admitted to the neurosurgical service of the University Hospital, Aug. 26, 1921, on Oct. 21, 1916, was wounded by a high explosive shell in the battle of the Somme. A diagnosis was made of compound fracture of the right ilium with sciatic nerve involvement. For four weeks following the operation the right leg was paralyzed and numb. He had had no sensation from the hip down. When sensation and motion began to return, the pain developed, soon becoming severe. He remained in army hospitals for eighteen months, the wound in the back healing one month before discharge. He returned to his home in Canada in May, 1918, suffering severe pain without any relief from medication. In May, 1919, he was operated on at the University Hospital (chordotomy), and for six weeks subsequent to operation he was free from pain, though conscious of a sensation in the foot, which was not annoying. The "real pain" returned about the end of July or August, 1919. The pain before the operation was referred to the thigh as well as the foot; in fact, it was more intense in the thigh than at any other place. Since the operation, when the pain returned, it was referred only to the foot and had continued there ever since; it began in the heel and extended along the sole to the big toe. The pain varied in intensity, but in some degree it was continuous, almost always confined to the foot, but occasionally felt in the lower part of the thigh, posteriorly.

Examination.—Aug. 31, 1921 (Dr. Spiller): Sensation: There was a distinct diminution of heat, cold and pain sensations in the right side of the abdomen below the level of the umbilicus, still greater in the anterior part of the right thigh and still greater on the inner side of the right leg. This impairment was present to about 1 inch above the umbilicus, but shaded off so gradually into the normal sensory area that a sharp definition was impossible. Tactile sensation was normal over the right side of the abdomen, anterior part of the right thigh and inner side of the right leg. It was greatly impaired down the outer side of the leg and dorsum and sole of the right foot.

The patient said that the pain was precisely as it was before the operation in both frequency and severity. It was evident that he had had a partial but not a complete interruption of the pain and temperature fibers from the right lower limb.

Reflexes: The left Achilles reflex and the left patellar reflex were normal. The right patellar reflex was weak. The right Achilles reflex was absent.

Operation and Course.—Sept. 20, 1921: Two spinous processes were exposed and isolated above the previous laminectomy, but only one of these, the lower one, was removed, together with the corresponding lamina. There had evidently been partial regeneration of the lamina next below. The special needle was introduced at a point on the left aspect of the cord midway between the origin of the anterior and posterior roots. As much tissue as the needle would contain was divided. There was no bleeding within the dural sac, with the exception of that which arose from the artery accompanying the posterior root at the level of section.

Sept. 21, 1921 (Dr. Spiller): On September 20, an attempt was made to cut one segment higher than at the previous chordotomy on the left side. As a result, the patient had complete loss of pain, heat and cold sensations in the anterior part of the right thigh and over the right side of the abdomen up to a line passing about 1 inch above the umbilicus. Here the differentiation between pain and cold was sharp, but for heat not quite so sharp. This level corresponds to the eighth thoracic segment. The patient had attacks of pain on the evening of the operation beginning in the right foot and not extending above the ankle. There was a sensation of constriction quite unlike anything he had ever had before, possibly the result of irritation of the central end of the fibers in the spinal cord. This sensation of painful constriction was not associated with any sensation of burning or coldness. The left lower limb felt heavy, but he moved it freely and wiggled the toes.

Tactile sensation was preserved in the anterior part of the right thigh and in the right side of the abdomen.

Sept. 26, 1921 (Dr. Spiller): On September 24, the patient had paresthesia in his left foot, which persisted, although it was not continuous. He could not describe the condition satisfactorily. The condition was not unlike the sensation produced by an insect crawling over the foot, but was not always of the same character. It was never painful. He still had complete loss of pain sensation to about 1 inch above the umbilicus on the right side. The pain, which had developed since the operation, persisted in the right foot; it was unlike any sensation he had ever had previously, but he said that if he never had any more acute pain, he could bear it easily. The pain about the chest, in a band just above the level of the operation, was probably due to the manipulation of the cord at the operation, and was slight.

Sept. 28, 1921 (Dr. Spiller): Paresthesia of the left lower limb had been less during the last twelve hours than at any time since September 23. This sensation had never been one of pain. The patient was entirely unable to describe it adequately. The sensation was not one of numbness, tingling, the feeling of insects crawling over the skin, or of the part "being asleep." The sensation of pain for pin prick and of heat and cold, objectively tested, was normal in the left foot, as in the left upper limb. Pain, heat and cold sensations were still completely lost to about 1 inch above the umbilicus on the right side. Spontaneous pain in the right foot had been less during the last twelve hours.

Oct. 5, 1921 (Dr. Spiller): The curious feeling of paresthesia in the left foot was much diminished, but the pain in the right continued practically unchanged.

Oct. 8, 1921 (Dr. Spiller): Heat and cold sensations were completely lost in the whole right lower limb and over the right side of the abdomen as high as a line about 1 inch above the umbilicus. Above this for about 2 inches there was a disturbance, but not loss of heat and cold sensations, on the right side only. This was true also of pain sensation. He did not feel a pin prick in the whole right lower limb and as high as a line about 1 inch above the umbilicus. Above this the sensation to pain was distinctly diminished for about 2 inches.

There was not the slightest impairment of motor power in the left lower limb, nor had there been any since the operation. Tactile sensation in the sciatic distribution of the right side was the same before the operation. The

right side of the scrotum had normal tactile sensation. The left side had normal sensation. The right side of the penis had diminished, not lost, temperature and pain sensations and normal tactile sensation. The left side of the penis had normal sensation. Sense of position was normal in the left big toe. The right big toe was affected by the old injury. The right patellar reflex was normal; the left was distinctly exaggerated, but there was no patellar clonus. There was persistent ankle clonus on the left side. The Babinski reflex was uncertain on the left side. There was no distinct movement on either side, although the movement seemed to be downward.

The patient had been walking frequently and had noticed no weakness in the left lower limb. He still had the same pain in the right foot that he had immediately after the operation. It had been of about the same intensity and frequency, possibly a little more frequent during the last week; it was very different from the pain he had had before the operation. It was not nearly as severe as the pain before the operation, but the frequency was about the same. A deep pin prick in the analgesic area was not perceived as pain.

Oct. 9, 1921: The patient was discharged.

Second Admission.—Feb. 20, 1922 (Dr. Spiller): The patient was readmitted to the hospital. After leaving the hospital following the second operation (chordotomy, September, 1921), the pain in the right foot gradually increased and finally involved the whole of the sole of the foot but not the dorsum. There was no spontaneous pain in the dorsum of the foot.

Until about three weeks before he had had no other pain; then he began to have pain about the middle of the posterior surface of the right thigh over the course of the sciatic nerve. The pain did not occur in the thigh unless he had pain in the right foot, and was present in the right thigh only when the pain in the right foot was intense; it seemed to radiate upward from the foot. He had some pain in the right foot all the time, but it was usually of a mild character, with an occasional exacerbation, severe enough to cause him great discomfort, but not as severe as the pain before the first chordotomy. While his sleep was disturbed by pain, he did not take drugs to control it; he admitted that his pain was decidedly less than before the first chordotomy.

Feb. 23, 1922 (Dr. Spiller): Over the dorsum of the foot and the sole on the right side all forms of sensation (touch, pain, heat and cold sensations) were lost. On the outer side of the right leg and down the posterior side of the right thigh, touch sensation was very indistinct, that is, throughout the distribution of the right sciatic nerve. Elsewhere in the right lower limb, and over the front of the right lower part of the abdomen, tactile sensation was evidently normal.

Heat and cold of extreme degrees (ice and very hot water) were not recognized anywhere in the right lower limb or over the right side of the scrotum, or on the right side of the abdomen to about 1 inch above the level of a line passing through the umbilicus. Pin prick was felt in the right lower limb only in an area on the inner side of the right leg, beginning quite close to the patella, bounded on one side by the crest of the tibia and on the other by a parallel line 1 inch to the inner side. This area widened as it extended to the heel, and terminated at the beginning of the sole of the foot. In the lower part of this area of preserved pain sensation, slight percussion with the finger caused a stab of pain. Deep pin prick anywhere else in the right lower limb, including any part of the thigh and the right side of the lower part of the abdomen, as high as the umbilicus, was not appreciated.

After sitting for some time there was momentary weakness in the left lower limb. No weakness of the left lower limb could be detected in resisting active movement to it. There was no ankle clonus on the left side. The left Achilles reflex was normal. The left patellar reflex was not unusually prompt, but it was a trifle more so than the right.

It would seem from this examination that the pain fibers from the lower and inner part of the right leg had escaped in the chordotomy, and this would

indicate that they were probably the more posterior or interior fibers of the anterolateral tract of the left side.

Second Operation and Course.—Feb. 24, 1922: A resection 6 cm. long was made of the saphenous nerve under local anesthesia. The nerve was exposed by a vertical incision after it had divided into its two terminal branches. Pinching one of these elicited pain precisely in the region in which the patient had been complaining of pain so bitterly, that is, in the inner side of heel. The branches were traced up to the main trunk where the upper section was made. After the resection, pinching the central stump elicited pain on the inner side of the foot and heel.

Feb. 26, 1922: The patient said that this was the first time in five years that he had been entirely free from pain or disagreeable sensations of any kind, except when under the influence of drugs. Before, when the acute pains were not present, a peculiar sensation had annoyed him. He could hardly believe that it was true, but he had had no pain or sensation in his foot for more than two hours on this date. His wife said that, to her knowledge, this was the first time in five years that he had been free from pain.

Feb. 27, 1922 (Dr. Spiller): When seen, on February 24, he was having severe pain in the toes of the right foot, probably as severe as before the operation, but possibly not as frequent. He had had a good night following the day of operation, and when seen the next morning (February 25) he was still suffering a great deal of pain in the toes of the right foot. He suffered great discomfort in the right foot all day.

He had had much better nights since February 25, and since then had felt remarkably better. He had practically no pain all day on February 26, having intervals as long as two hours without the slightest twinge of pain or sensation of any sort in the right lower limb. After an hour or two he might have a twinge of pain with drawing up of the right lower limb. It was never severe and of only a few minutes' duration. He went to sleep the evening of February 26 "like a child," the first time since the injury.

On February 27, there was slight pain about the middle of the side of the right foot. On February 25, the intense pain of the preceding night was believed to have been caused by the irritation of the central stump of the long saphenous nerve after section, the nerve not yet having lost its function. He had never felt so encouraged before.

March 7, 1922 (Dr. Spiller): The patient said that the pain was over the whole of the sole of the right foot, but that it was more intense over the inner half than over the outer half.

Third Operation and Course.—March 11, 1922: A laminectomy and chordotomy was performed on the left. Section was made, 3 cm. deep, at the level of the tenth thoracic segment.

March 12, 1922 (Dr. Spiller): Touch, pin prick, heat and cold apparently were normally recognized throughout the whole left lower limb. He did not move the left toes freely on account of suffering from the operation of the previous day. No further examination was made. The day and evening of the operation, he had some pain in the right toes, which continued. It was located chiefly in the right toes. Before the operation it seemed to be chiefly in the sole.

March 14, 1922: The patient had his first bed bath on the morning of this date. While being washed on the outside of the left thigh, he had a peculiar sensation similar to that which he had had for years in his right hip. It was almost a numb sensation. He felt the pin prick as distinctly in the right as in the left thigh. Occasionally he seemed to feel the pin prick a little less acutely in the left thigh.

Heat and cold sensation, however, were distinctly impaired in the left thigh. He repeatedly called the ice cold tube "hot" at any part over the thigh, although sensation was a little more acute over the posterior surface. He

could not readily appreciate heat in the same region. Below the left knee, temperature sensation, though impaired, was not lost.

The patellar and Achilles reflexes on the left side were normal. There was no clonus on the left side. The right patellar reflex was absent. The Babinski test gave no distinct flexion of the left toe. There was no weakness of the left lower limb.

The patient had a little less pain during the morning of March 14 than before. It came in paroxysms.

March 16, 1922: The sensation of pin prick in the left thigh was fully as sharp as in the left upper limb. The same was true of tactile sensation. During the previous night and the morning of March 16 he had "scarcely any pain." Heat and cold sensations were practically lost over the front and on each side of the left thigh, but only a little, if at all, impaired, over the back of the thigh.

Sensation for heat and cold was greatly impaired over each side of the abdomen as high as the nipples (that is, the fourth intercostal space). Pin prick sensation was lost to about 3 inches (7.62 cm.) above the umbilicus. The loss of heat and cold sensation on the left side of the trunk up to the fourth interspace suggested temporary injury of the right side of the cord sustained during the operation.

March 20, 1922 (Dr. Spiller): The patient complained of considerable "burning" pain in the whole sole of the right foot. It was, therefore, associated with a sensation of heat. He said that he had been subject to muscle cramps all his life, but this pain was not of the same character. All the pressure made with the hand in grasping the limb produced no pain in the right thigh or foot. The spontaneous pain, therefore, apparently was not in the deep peripheral sensation.

March 22, 1922: His tactile sensation and pressure sensation were acute in the right thigh, but all the pressure that could be brought to bear with a fountain pen over the femur of the right thigh produced a distinct sensation of pressure, but not the slightest discomfort; whereas a moderate amount of pressure over the left thigh produced discomfort, and then pain. Vibratory sense was normal and equal as tested over each patella.

March 28, 1922 (Dr. Spiller): Sensation to touch and pin prick was normal in the thigh and leg on the left side and was equal in intensity with the sensation of the left upper limb. There was a distinct impairment in heat and cold sensations in the left lower limb and left side of the abdomen to a level of 2 inches below the nipple. In this area, ice-water was repeatedly called "hot." Very hot water in this area was always recognized correctly as "hot." Careful testing of pain sensation in the left thigh and leg failed to elicit any disturbance of pain sensation. The peculiar paresthesia which he had noted in the left thigh previously when being bathed, had entirely disappeared.

March 29, 1922 (Dr. Spiller): The patient's wife said that after the first operation, May 24, 1919, he had had no pain of any consequence for three months, and every operation had been followed by relief of pain for an average of three months, although the relief had not been absolute. After the second spinal operation a trip to Florida caused great improvement in his condition. He had less pain while there. When he was in the company of strangers, he seldom showed pain. There evidently was a psychic element in his pain. He was much more likely to have pain when alone with his wife than when with others, at which time, if any argument arose, the pain immediately increased.

March 31, 1922 (Dr. Spiller): His nurse said that for four nights and three days the only medicine he had received for relief of pain was a capsule of sugar, and this had always given relief. He had asked for it again when pain had recurred. The only drug he had received to make him sleep during the two previous nights had been a capsule of sodium bicarbonate, and he had slept as well after taking this as he did after taking barbitol. This statement may be offered as evidence that his pain had a large psychic element.

April 1, 1922: The patient was discharged.

June 12, 1922: The patient reported that the pain was still present in the sole of the foot; it was only occasionally present in the lower part of the thigh and was not as severe as formerly. There was no abnormal sensation in the "good" lower limb; but when he wakened in the morning, or after sitting for some time, he could not use this limb, possibly for half a minute.

CASE 8.—*History*.—E. S. S., Jr., aged 40 years, referred to the neurosurgical service of the University Hospital by Dr. Hugh T. Patrick and Dr. T. J. McKinney, said that he had been entirely well until 1 p. m., May 12, 1921. While attending a committee meeting, he began suddenly to have severe pains in the back about the lumbar region. The pains came in waves, and at first he thought them due to his bowels. An hour later the pains became so severe that he became pale and weak. He went to a physician's office. The physician thought there was some intestinal disturbance, and gave him a drastic purge. He said he told the physician that he felt as though he had lumbago, and he rested in the physician's office for a time. Later he walked home with difficulty, and neighbors told him that his limbs seemed to drag. He noticed that his legs seemed to be going to sleep, and he was unable to use them. When his wife returned she assisted him to bed; he was able to lift the left leg up on the bed, but not the right. The next day he felt a definite girdle sensation around the waist, about at the level of the umbilicus. About two weeks later he had another attack, which consisted of severe pains in the back, radiating around to the sternum just below the nipple line. Later the girdle sensation disappeared, and he gradually lost sensation up to the nipple line. The second attack seemed to involve the neck and arms. He was unable to use the arms for about two weeks. The eyes required subdued light. The level of the lesion had remained the same as that after the second attack, and he had had no further "attacks." There had been a constant pain, dull in character, in the right side and in the back. During the first attack, there was some febrile reaction, which continued through the second attack.

Since the second attack there had been a band of hyperesthesia about the chest. This was so painful in the right axilla that it was necessary to prevent contact over this area. The bed clothing was not bearable, and he held it off with a frame or with his arm. Since the first attack he had lost all motor and sensory function below the waist. He had been incontinent in regard to both urine and feces. During the last two months, he had had five attacks of hematuria, lasting from three to seven days.

His brother, a physician, said that he and the patient were "bleeders," and that slight cuts caused quite severe hemorrhages. The patient asserted that he had had no serious hemorrhages, except after the extraction of a wisdom tooth.

Physical Examination.—Head, neck, heart and lungs were normal. Decubitus was present over the buttocks on each side, and there was one sore above the coccyx.

Lower Extremities: There was absolute flaccid paralysis of both lower extremities. There was loss of tactile pain and temperature sense below the level of the sixth rib.

June 5, 1922 (Examination by Dr. Spiller): The patient's wife said that for two months after the onset of his symptoms he had had no hyperalgesia. Hyperalgesia began about the end of August, 1921. The condition of the lower limbs was one of complete flaccid paralysis, with much atrophy and complete loss of sensation for touch, pain and probably all other qualities. He complained of a burning sensation in the right knee; he felt this sensation occasionally in the left knee. The pain about the chest was much more severe on the right side, and extended from the spinal column all the way to the midline of the chest. The pain was in a band about 2 inches wide; the upper border of which was at the level of the nipple. The pain in the left side was

only in front and extended about 1 inch above the nipple. It was stinging, burning or throbbing, always present, sometimes with severe exacerbations, and worse at night. The zone of irritability to touch was wider than the zone of spontaneous pain. The zone of hyperirritability to touch began on the right side at about the third intercostal space, and extended to about 2 inches below the right nipple. It was about the same in width over the left side of the chest. When this zone was touched, the patient winced and showed pain; even touching the hair in this area caused a stinging sensation—not sharp pain. There was no increased sensitiveness to touch or pin prick in either axilla. The pupils were equal and normal in size. There was no widening of the palpebral fissures and no protrusion of the eyeballs, that is, no evidence of ophthalmic sympathetic involvement. The patient said that the hypersensitiveness of the chest had been getting worse during the last three or four months.

RESULT OF TESTS OF REFLEXES

Reflexes	Right	Left
Biceps	++	++
Triceps	++	++
Abdominal	0	0
Patellar	0	0
Achilles	0	0
Babinski	0	0
Clonus	0	0

Urinalysis was negative. Blood examination revealed that the clotting time was six and one-half minutes. The spinal fluid pressure was 10 mm. of mercury, and the spinal fluid contained four cells; it was negative for globulin and sugar. The Wassermann blood and spinal fluid tests were negative. Roentgenologic examination was negative.

Operation and Course.—June 7, 1922: An anterolateral section, bilateral, at the level of the first thoracic segment was made. Beginning under local anesthesia, the spinous processes and laminae of the seventh cervical and the first thoracic vertebrae were removed. The oozing from the margins of the laminae and from the muscle surface was unusually stubborn, and continued so throughout the operation. Therefore, the field was not nearly so dry as we would have wanted to have it before the dura was opened. When the dura was opened it was found that a posterior root on the left side, presumably either the last cervical or the first thoracic, was given off exactly in the middle of our laminectomy opening and just opposite our guide suture, which corresponded to the tip of the seventh cervical vertebra. As the laminectomy opening was small, it was rather inconvenient to have the posterior root given off exactly in the middle of the opening, but with a silk suture it was gently retracted upward, and the root section was made immediately below. Right Side: The root on the right side came off at a slightly lower level. It was retracted gently downward, and the chordotomy section was made just above it. It was noted that throughout the operation when the patient complained of pain it was referred to the arms. The dural incision was closed and considerable time was spent in trying to control the oozing from the various points in the wound; after one point was closed another would begin to ooze, so that we became rather discouraged, although with the aid of muscle grafts and warm tampons we were finally able to secure a sufficiently satisfactory hemostasis to warrant our closing the wound. This was done with interrupted tier sutures of silk. A Mickulicz drain was introduced in the lower angle of the wound. Convalescence was uneventful. The wound healed by first intention.

June 8, 1922 (Examination by Dr. Spiller): Both pupils were very small, the right smaller than the left. The palpebral fissures did not seem to be as wide as normal, and the right was not quite as wide as the left. He had spontaneous pain in the hypothenar eminence of the right hand, and while pin prick was felt here as sharp, it was not quite as acute as it was over the right thumb. He had, therefore, symptoms indicative of ophthalmic sympathetic involvement, more on the right than on the left. Morphia may have been partly responsible for the contracted pupils. It hardly seemed to explain the whole finding because of inequality of the symptoms on the two sides and because of the spontaneous pain and slight hypalgesia in the distribution of the first thoracic nerve. The grasp in the right little finger did not appear to be quite as powerful as the grasp in the left little finger.

June 9, 1922 (Examination by Dr. Spiller): In looking straight forward without making any special effort, the palpebral fissures were distinctly subnormal in width, the right more so than the left. In making an effort to open the eyes wide the fissures were wider, but still subnormal. The right pupil was a little smaller than the left. The patient had complained during the past twenty-four hours of considerable pain and numbness down the inner side of each forearm and in the ring and little finger of each hand, more in the right than in the left. The spontaneous pain and hyperalgesia to pin prick over the upper part of the thorax had disappeared entirely.

June 27, 1922 (Examination by Dr. Spiller): The right pupil and the right palpebral fissure were distinctly smaller than the left, and the right eyeball did not protrude as much as the left. When asked to open his eyes, or to look upward, the palpebral fissures were about normal. The reaction of each pupil to light and in convergence was normal. The flexor power of each little finger was still very weak. The pain that was present after the operation on the ulnar side of each forearm and hand had entirely disappeared, but he still complained of numbness—of less intensity than immediately after the operation—in the little and ring fingers of each hand and extending up the ulnar side of each forearm and corresponding part of each arm as high as the axilla. There was diminution in touch, pain, heat and cold sensations, but no loss on the ulnar side of the left forearm, in the corresponding part of the left arm above the elbow or in the ulnar side of the left hand. This disturbance of the same character was greater in the right upper limb than in the left and was in the ulnar side of the right forearm and also the corresponding part of the right arm above the elbow. In the ulnar side of the right hand just as in the ulnar side of the left hand, touch, pain, heat and cold were felt as well as in the area of the thumb. This greater loss of objective sensation in the right side corresponded to the greater sympathetic palsy of the right side, and with the numbness in the same parts and the previous pain in these parts was indicative of root rather than cord damage. Sense of position and passive movement was normal in each little finger and wrist. Vibration was normal on the ulnar side at the wrist and elbow. Hyperesthesia to the slightest contact, even of such extent as to produce dread of an object like a cloth being held near the trunk, had entirely disappeared from the upper thoracic region above the area corresponding to the level of the lesion. Within the last two days he had complained of a slight discomfort from contact on the right side in the upper thoracic region, worse at night, when his mind was not diverted, and at no time of much importance. Sensation for touch, pain, heat and cold was impaired but not lost from about the second rib to about the fifth intercostal space on each side, and they seemed to be a little more impaired on the left side of the trunk; but this was not entirely certain. It is to be remembered that the sympathetic fibers for the eye probably leave the cord through the anterior first thoracic root. The objective disturbance of sensation corresponded more to the second thoracic distribution.

Summary: Following section of the anterolateral columns on both sides at the level of the first thoracic segment, the distressing hyperesthesia complained of before the operation disappeared. There were transitory pains in the upper extremities but these gradually disappeared. An area of hypesthesia persisted along the ulnar aspect of the forearm and hand of each side. Following the operation sympathetic ocular phenomena were observed.

DISCUSSION

DR. BERNARD SACHS, New York: May I ask for a little additional information regarding the kind of cases in which operation was performed? Were they cases of tumor somewhere in the spinal cord or in the spinal canal? Moreover, was this attempt made in cases of tabes, and what was the nature of the case in which the pain was so extreme that the necessity arose to operate on the patient for the diminution of the pain?

Was the operation undertaken without any attempt to remove the tumor in those cases in which there was clearly a spinal tumor?

I have seen extremely few cases of malignant or other disease of the spinal cord in which the pain was so pronounced that I would have considered a serious operation for the relief of pain.

DR. M. ALLEN STARR, New York: I should like to ask whether subsequent to this operation any trophic disturbances appeared in the areas of the skin which were deprived of pain and temperature sense. We are familiar with the fact that in cases of syringomyelia such trophic disturbances occur, which have been ascribed partly to the loss of pain and temperature sense.

DR. SMITH ELY JELLIFFE, New York: Dr. Starr's question leads me to ask another one in which physiologic problems may enter the field of discussion. Did Dr. Spiller or Dr. Frazier make any inquiries as to the possibilities of metabolic pathways traversing in the anterior columns? Were finer tests of the kidney or liver functions investigated? Was there any evidence to show that some of the pathways that are utilized in the integration of visceral function, such as the experimental work of Brugsch, Dresel and Lewy² have shown, might pass up in that region.

DR. CHARLES H. FRAZIER, Philadelphia: The value of the communication by Dr. Spiller and myself seems to lie in the physiologic results or observations which have been drawn from the cases in which we have operated.

Two or three years ago I reported to this Association the results of the first series of chordotomies which I had performed in the neurosurgical clinic of the University Hospital, and I was quite confident at that time that the results of the operation justified the undertaking, in so far as the measure of relief was concerned. In its conception, chordotomy seems to be quite comparable to Dr. Spiller's original suggestion for avulsion of the sensory root of the gasserian ganglion in the treatment of trigeminal neuralgia, and in their effect the two operations are quite similar.

With regard to the technic: One operation is easier of performance than the other; since in avulsion of the sensory root the whole root must be sacrificed, whereas in the chordotomy the operator must exercise his judgment as to the amount of chord tissue to be sectioned. We know that in different persons

2. Dresel and Lewy: Die Zuckerregulation bei Paralysis Agitans Kranken. Ztschr. f. d. ges. exper. med. **26**:95, 1922. Die Widal Leberfunktionsprüfung bei Paralysis Agitans Kranken, *ibid.* **26**:87, 1922. Jelliffe and White: Diseases of the Nervous System, Ed. 4, 1922.

the size of the cord varies and even in the same person the size of the cord varies at different levels. It is for that reason that we have thought it advisable to recommend section of the cord at a given level, and for various reasons we recommend that the section be made at the level of the fifth thoracic segment.

Those who contemplate the performance of this operation should provide themselves with two special instruments. One is a hook which we have devised for the purpose of grasping and including only the anterolateral column of the cord and fixing the tissue so that the section may be made without undue trauma; the other is a special knife, so constructed that the section may be made without any technical difficulty, the blade being at an obtuse angle to the shaft.

DR. ERNEST SACHS, St. Louis: I have had opportunity to perform this operation on three or four patients, two of which were tabetics. The first patient was not relieved, this probably having been due to the fact that the section was not performed high enough.

The most satisfactory case I have had was one of carcinoma of the prostate, with metastasis in the hip joint, a case which seemed hopeless. The man was in excellent physical condition and would have been able to walk around had it not been for his intractable pain. Operation in this case relieved the patient completely, so that he was able to resume his work. He is still carrying it out six or seven months after the operation, but it is difficult to state how long he will continue to be relieved.

In the last case I made use of Dr. Frazier's hooks, and they unquestionably make the operative procedure very much simpler. I think the operation has a great field of usefulness in cases of carcinoma with metastasis to the spine, but I should hesitate to operate in tabetic patients.

DR. SPILLER, in closing: In reply to Dr. Sachs: We mentioned that in four of these cases there were malignant growths, definitely shown to be malignant either by operation or by the roentgen-ray findings. In two there were injuries received in France, with intractable and intense pain as a result, and in one, a woman, there was intense pain of long duration possibly from disease in or about the bladder. These were suitable cases for chordotomy. Of course, it would be an entirely unjustifiable procedure to perform a chordotomy if there were a possibility of removing a tumor; and we have never attempted it if there was the slightest chance of removing a tumor from the cord.

We have had no cases of tabes in this series.

Dr. Sachs says he has not seen many cases of severe pain in malignant tumor. We have observed most intense pain in malignant tumor of the vertebral column and great relief after chordotomy.

In reply to Dr. Starr's question whether there were trophic disturbances after the operation: We have not observed any changes in the skin or in the growth and appearance of the nails, but in two cases bedsores resulted. In one of these there was unquestionably implication of the crossed pyramidal tracts, while in the other the symptoms did not indicate such implication.

In regard to Dr. Jelliffe's question whether metabolic pathways were studied: We have not made a careful study of these, nor have we observed anything resulting from this operation that might point toward metabolic changes. One might expect hematuria possibly, but there was nothing of that kind in any of these cases; there was nothing that called our attention to any disturbances of the viscera.

CLINICAL NOTES ON THE PATHOLOGY IN A CASE
OF EPIDEMIC ENCEPHALITIS COMPLICATED
BY A PSYCHOSIS *

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While instances of epidemic (lethargic) encephalitis complicated by various psychoses are by no means rare, studies of the pathology of such cases, except those of so-called striatal and pallidal syndromes, are exceedingly scarce.

REPORT OF A CASE

History.—A colored woman, 28 years of age, admitted to the Cook County Hospital on Jan. 7, 1921, complained of pains in the arms and shoulders, tremor of the left upper extremity and occipital headache. The pain was "stinging," shooting from the shoulders, especially the left, down to the fingers, while the tremor was of the "intention" variety. Both the pain and the tremor were of six days' duration. The headache dated back to an attack of influenza about eight months before.

Physical Examination.—Examination revealed jerking and tremor of the left upper extremity, at times also of the right, and marked hyperesthesia of the neck, arms and chest. The heart, lungs and genito-urinary organs were normal. The temperature was 99.8, the pulse 82, respiration 24. The spinal fluid showed 20 cells (small lymphocytes), no globulin and a negative Wassermann reaction.

Course of Illness.—About a week later there was internal strabismus; the patient complained of diplopia vertigo and "ringing in the head." Twitchings in the left side of the face, drowsiness, hallucinations of vision and a masklike face appeared. The pupils were equal with sluggish reaction to light and absence of reaction in accommodation; there was slight ptosis of the left lid, but the strabismus disappeared. The temperature, which had ranged between 100.4 and 102 F., became normal, and the patient, feeling quite comfortable, left the hospital on March 8, two months after admission.

Readmitted about two weeks later, she was noisy, violent, restless and resistive. She was transferred on April 1, 1921, to the Psychopathic Hospital and thence the next day to the Chicago State Hospital, where she died on Jan. 22, 1922.

During the two weeks at home she had developed a somewhat indefinite psychosis. She was for the most part in fear of devils and snakes; believed that she was to be poisoned; was prayerful; thought she was dead and in another world. She was confused and attempted to jump through a window;

* From the Pathology Laboratory of the Illinois State Psychopathic Institute.

she also made a violent attack on her husband and children. But she had a short period of parietic-like expansiveness in which she believed that she and the entire family were going to visit relatives in the South, and that she was going to do everything in grand style; she wanted all of them to have new outfits of clothing, etc. The money was to come from a drawer which was filled with thousands of dollars, placed there by God. God was telling her at that time that she could have whatever she wished.

On admission the neurologic picture had faded, a somewhat sluggish pupillary reaction and some bilateral divergent strabismus remaining. She presented a picture of extreme episodic excitement and proved to be a most violent patient. Although she was a very small woman, it was frequently necessary to use physical restraint. She would often attack any one within reach and appeared to be unclear at the time of these attacks. The only thing that her excitement can be likened to is the extreme furor of an epileptic. She would dash through a window, cutting herself severely, just as an epileptic does.

At other times her reactions were of quite a different kind. She would cry, ask forgiveness for her conduct, and beg those about her to be good to her. At these times her contact with environment was apparently good. She soon learned the names of all who came near her and would even pick up their first names. She was in an unquestionably delirious state at times, when she would confuse those about her with members of her own family. At no time was her stream of speech a clear one, nor could it ever be actually directed.

She remained in practically the same condition for eight months, during which time her general physical condition remained stationary. Then her excitements became more intense and more frequent. Finally, during these excitements the patient began, without explanation, to mutilate herself. One of the results of this self-mutilation was the production of a rectovaginal fistula. Attempts to treat this fistula proved ineffective because of the interference of the patient. On Jan. 17, 1922, her bed was found covered with blood. She had succeeded in pulling a loop of intestine through the fistula, and there was a complete tear through the intestine. She did not complain and made no explanations, exhibited no signs of shock and did not seem distressed. She was immediately operated on and died two days later with peritonitis.

Histopathologic Report.—Microscopic examination of the apparently normal brain revealed the following:

Ganglion Cells and Nerve Fibers: Many ganglion cells, especially in the cortex, appeared quite normal. Some showed marked destruction; some neuronophagia, chromatolysis or cell sclerosis, especially in the basal ganglions and midbrain. Throughout the cortical layers which retained their general architecture, as well as in the granular layer of the cerebellum, in the basal ganglions and midbrain the nerve cells exhibited an enormous accumulation of lipoids. Lipoids were also found in the ependymal cells of the aqueduct of Sylvius and of the lateral ventricles. Especially abundant were lipoids in the optic thalamus, substantia nigra, pons and corpus striatum, while the globus pallidus, nucleus ruber and the occipital lobe showed comparatively little fat in the cells, but large amounts in the blood vessels. The nerve fibers, the axons and myelin showed no changes whatever.

Glia: Proliferation of glia nuclei was quite marked in the deeper strata of the cortex, also in the optic thalamus, putamen and the region of the aqueduct. In the latter areas, as well as in the nucleus dentatus cerebelli, there were numerous cytoplasmic glia cells, some containing two nuclei, and many rod

cells (Stäbchenzellen) considered by some gliogenous, by some mesodermogenic elements. Stained with scarlet red the glia cells revealed minute droplets of lipoids.

Blood Vessels: In the midbrain and the basal ganglions (especially in the optic thalamus) some vessels were mildly infiltrated with hematogenous elements (lymphocytes, plasma cells). In other places the dilated Virchow-Robin spaces contained vacuolated bodies filled with yellow granules (in specimens stained with toluidin blue or methyl blue eosin). Such vacuolated bodies much resembled gutter cells packed with lipoids, and were also found in the cortex (especially in the occipital region). Much more marked were so-called proliferative or productive phenomena. Mild and unusual in the cortex, they were much in evidence in the subcortical regions, and especially in the nucleus caudatus, pons and around the aqueduct, where they showed as hypertrophied deeply stained endothelial cells, buddings and new formed capillaries. Much more common and widespread were infiltrations of the small and larger vessels with lipoids which in some instances (basal ganglions and midbrain) were present in large amounts.

Pia-Arachnoid and Choroid Plexus: The pia-arachnoid was markedly infiltrated with mesothelial cells, fibroblasts, some lymphocytes and modified plasma cells. Over the occipital and parietal lobes the distended meshes of the pia-arachnoid contained typical ganglion cells, some surrounded by lymphocytes, some perfectly intact, with a distinct nucleus and processes. Such a peculiar phenomenon observed by one of us (G. B. H.) in many other conditions is recorded here as a curiosity. This heterotopia evidently was an artefact. The arachnoid villi (pachionian bodies) were greatly hypertrophied, hyperplastic. The tuft cells of the choroid plexus contained an abundance of large granules and vacuoles, a well developed nucleus rich in chromatin, while the perivascular stroma was densely infiltrated with cell bodies, some of which much resembled mesothelial or endothelial cells. Both the pia-arachnoid and the choroid plexus contained large amounts of lipoids.

Summary of the Microscopic Findings.—1. Mild inflammatory infiltrative phenomena in the basal ganglions and midbrain.

2. Widespread proliferative or productive vascular changes throughout the brain—pronounced in the basal ganglions and the midbrain, but rare and mild in the cortex.

3. Diffuse parenchymatous changes with a vast accumulation of lipoids in the nerve cells, blood vessels, choroid plexus, pia-arachnoid and to a lesser extent in the glia.

4. Proliferation of glia in the basal ganglions.

COMMENT

We have in this case a combination of diffuse inflammatory and degenerative phenomena. Both were especially in evidence in the basal ganglions and midbrain. But the infiltrations were not so pronounced as in acute cases of epidemic encephalitis, while the proliferative changes were not so marked as in so-called productive encephalitis (caused, for instance, by lead or arsenic). Resembling in the combinations of both inflammatory and degenerative changes the findings in general paralysis of the insane, this case differs from the latter in the comparatively mild involvement of the cortex. The inflammatory

changes in the basal ganglions and midbrain could perhaps explain the nervous manifestations in our patient (ocular paralysis, parkinsonian face), while the diffuse degenerative phenomena in the cortex were most likely responsible for the patient's mental condition. It is obvious that degenerative lesions are a serious complication of epidemic encephalitis, much more serious than the inflammatory changes. For the latter may subside or disappear entirely, leaving no permanent disability, in contrast to a degenerative process, which, however mild, may produce more or less troublesome complications of which the most dangerous are mental disturbances.

As the cause of the degenerative changes is most likely toxins elaborated by the virus of epidemic encephalitis, and as they may prove very virulent and dominate the pathologic condition, it follows that the prognosis of epidemic encephalitis is always doubtful.

In conclusion we wish to call attention to a purely histopathologic feature: fat accumulation in the ependymal cells of the lateral ventricles, in the pia-arachnoid and tuft cells of the choroid plexus. This has been discussed by one of us (G. B. H.) in previous contributions and is pointed out only to emphasize the fact that such fatty infiltrations occur in any degenerative condition.

NEUROSYPHILIS AMONG THE CHINESE

WITH FINDINGS IN SIXTY-FIVE CASES

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INCIDENCE OF SYPHILIS

Practitioners in China agree that syphilis is prevalent among the Chinese. In order to secure statistical information on the subject, a request for annual reports was made of all hospitals in China. Of the reports secured, twenty-nine from fifteen different hospitals¹ contained lists of diagnoses made on inpatients. Three of these hospitals in addition published diagnoses made on outpatients.

For comparison, data from the annual reports of nine hospitals² in America was compiled. Statistics from these two groups of hospitals are presented in Tables 1, 2 and 3. Lack of uniformity in diagnostic

TABLE 1.—INCIDENCE OF SYPHILIS AMONG HOSPITAL PATIENTS
IN CHINA AND AMERICA

General Hospitals*	Inpatients			Outpatients		
	Total Number of Patients	Number of Cases of Syphilis	Percentage of Cases of Syphilis	Total Number of Patients	Number of Cases of Syphilis	Percentage of Cases of Syphilis
China.....	35,525	2,936	8.4	57,242	3,513	6.1
America.....	100,143	2,516	2.5	68,402	2,063	3.1

* In these tables data concerning inpatients are from fifteen hospitals in China and nine in America, and concerning outpatients from three hospitals in each country. Patients in all departments are included.

facilities, in disease classifications used and in methods of compiling medical statistics in the various hospitals, does not permit a close comparison of figures.

It is evident from Table 1 that the incidence of syphilis among the patients of the hospitals selected is two or three times greater in China than in America.

1. The hospitals, with the number of inpatients reported (often for several successive years) are: Peking Union Medical College, 7,138; Canton Christian College, 6,976; Rankine Memorial, Ichang, 5,297; University of Nanking, 2,755; St. Luke's, Shanghai, 2,047; Church General, Wuchang, 2,017; Red Cross, Shanghai, 1,711; Wuhu, 1,393; Soochow, 1,384; Kaifeng-Hunan, 1,348; Chefoo-Shantung, 1,095; Shantung Christian College, 1,003; Tsing Wang Pu, 789; Siangtan, 560; Pei Yang, Tientsin, 318.

2. The hospitals, with the number of inpatients reported are: Johns Hopkins (whites), 6,151; Bellevue, 50,403; Presbyterian, New York, 7,324; New York, 5,944; Lakeside, Cleveland, 5,929; Peter Bent Brigham, Boston, 3,533; Pennsylvania, 5,662; University of Pennsylvania, 6,863; Massachusetts General, Boston, 8,834. The last three gave diagnoses also of outpatients.

The results of routine Wassermann tests performed on Chinese inpatients have been reported as follows: In Peking, of 494 male medical patients, 24 per cent. gave positive tests (Sia³). In Soochow, of 752 medical and surgical patients, three fourths of whom were male, the corresponding proportion was 39 per cent (Snell⁴). Similarly compiled statistics have been reported from many hospitals in America. The results of these examinations, summarized by Vedder,⁵ show from 10 to 30 per cent. of patients giving positive Wassermann reactions.

Wassermann tests performed in Peking on 800 Chinese domestic servants preliminary to employment yielded 11 per cent. positive tests (Korns⁶).

INCIDENCE OF NEUROSYPHILIS

In spite of the prevalence of syphilis among the Chinese, cases of neurosyphilis are apparently comparatively rare. The following testimony on the subject is given by men who have practiced among the Chinese for many years. Hodge⁷ saw only one or two cases of tabes dorsalis and one case of general paresis. Maxwell⁸ never saw a typical case of general paresis and only three of tabes. Reed⁹ saw only one or two cases of tabes.

Similar evidence concerning another branch of the Mongolian race is given by Montel,¹⁰ who found only one case of tabes among 100,000 patients treated in Indo-China. Personal application for information similar to that secured from hospitals in China and America was made of twenty of the leading general hospitals in Japan. It was found, however, that these hospitals do not keep a tabulated record of the diagnoses made. Thom¹¹ says that in Japan neurosyphilis is rare, but he does not cite the evidence on which the statement is based.

The comparative incidence of neurosyphilis in the two groups of hospital patients in China and America is shown in Table 2. Central nervous system involvement among syphilitic inpatients is seven or eight times more frequent in America than in China. The difference among

3. Sia, R. H. P.: Routine Wassermann Test on 502 Inpatients, China M. J. **35**:39 (Jan.) 1921.

4. Snell, J. A., and Chang, P. Y.: Report of Routine Wassermann Test at Soochow Hospital for One Year, China M. J. **35**:36 (Jan.) 1921.

5. Vedder, E. B.: Syphilis and Public Health, Philadelphia, Lea and Febiger, 1918, p. 97.

6. Korns, J. H.: Further Statistics on Communicable Diseases Among Domestic Servants, China M. J. **35**:382 (July) 1921.

7. Hodge, S. R.: Syphilis in China, China M. J. **21** (Sept.) 1907.

8. Maxwell, James: Syphilis Among Chinese, China M. J. **27** (Nov.) 1913.

9. Reed, A. C.: Nervous Diseases in China, China M. J. **29** (Nov.) 1915.

10. Montel, L. R.: Un cas de tabes chez un Annamite, Bull. Soc. méd. Chir. Indo-China, October, 1916.

11. Thom, B. P.: Strain in Spirochetes, Am. Jour. Syph. **5**:1 (Jan.) 1921.

outpatients is not so great because most of the outpatients in China are reported by one hospital in Peking, and this hospital, for reasons to be explained, treats a relatively high proportion of cases of neurosyphilis.

Additional evidence concerning the incidence of neurosyphilis is furnished by institutions for the insane. The Robert G. Kerr hospital at Canton is the only modern hospital for the insane in China. During the last seven years, 312, or 14.7 per cent., of the patients treated were diagnosed as having general paresis. Samuels¹² found only 3.4 per cent. among the Chinese in one asylum in Malay. The annual reports of thirteen hospitals¹³ for the insane in America, with admissions totaling 5,955, show an incidence for general paresis and taboparesis of 6.7 per cent. London Insane Asylums¹⁴ show a ratio for general paretics of 8 per cent., the New York State hospital a ratio of 18 per cent. for men and 13 per cent. for women (Kindred¹⁵).

TABLE 2.—INCIDENCE OF NEUROSYPHILIS AMONG HOSPITAL PATIENTS IN CHINA AND AMERICA

General Hospitals	Inpatients			Outpatients		
	Number of Cases of Neuro-Syphilis	Percentage of Patients	Percentage of Cases of Syphilis	Number of Cases of Neuro-Syphilis	Percentage of Patients	Percentage of Cases of Syphilis
China.....	150	0.4	5.1	90	0.16	2.7
America.....	1,003	1	39.8	159	0.23	7.6

Hofman,¹⁶ Woods¹⁷ and Harvey¹⁸ have presented the general physical and mental findings in some of the Robert G. Kerr hospital cases. Unfortunately, examination of the patients at this hospital has not included the Wassermann test of the blood serum or examination of the spinal fluid.

The bulk of the evidence at hand points to the comparative rarity of syphilis of the nervous system in the yellow race. Similar reports

12. Samuels, W. F.: General Paresis in the Tropics, *Indian Med. Gaz.* **51**:453 (Dec.) 1916.

13. These hospitals include eight state and one private hospital in Massachusetts, and one state institution in Connecticut, Maryland, Indiana and New Hampshire.

14. Report of the Royal Commission on Venereal Diseases, 1916, p. 137.

15. Kindred, J. J.: Venereal Diseases, Their Relation to Insanity and Nervous Diseases, *New York M. Rec.* **93**:184, 1918.

16. Hofman, J. A.: *China M. J.* **27** (Nov.) 1913.

17. Woods, A. H.: Diseases of the Spinal Cord Among the Chinese, *China M. J.* **32** (March) 1918.

18. Harvey, I. L.: Cases of General Paresis in China, *China M. J.* **34** (March) 1920.

concerning the black race have been made by several observers. At least five explanations of this apparent racial discrimination have been advanced:

1. Among the colored races there is a specific strain of spirochete which spares the central nervous system. Hofman¹⁶ says that many of his cases of general paresis in Canton occurred in steamship employees, who derived their infections from a non-Chinese source. Most of the hospitals in China reporting cases of neurosyphilis are located in coast cities which have long been exposed to western syphilization. More statistics are needed from inland China before the value of this argument as regards Chinese can be judged.

2. The colored races have a nervous system less sensitive and less susceptible to syphilitic infection than has the white race.

3. Among the colored races syphilis is a lately acquired infection and has not yet reached the attenuated form in which central nervous system damage is common. This argument would not hold for the Chinese because syphilis has been with them in all probability for many hundreds of years.

4. The colored races have not been subjected to modern antisymphilitic treatment, which, according to Fraser¹⁹ and others, leaves the spirochete, bandit-like, in the inaccessible fastnesses of the central nervous system. A possible illustrative case in our series is that of a Chinese cook for an American family, who on initial examination presented a chancre scar and a secondary eruption. He was given nineteen intramuscular injections of mercury and three intravenous injections of arsaminol, a Japanese preparation of arsphenamin. Six months after beginning treatment he became completely deaf. His ear drums were normal. Wassermann tests of the blood and spinal fluid were negative, but the spinal fluid gave a positive globulin test and contained 270 cells per cubic millimeter. The patient accepted discharge from his position rather than undergo any further treatment at the hands of the foreign physician.

5. The reputed low incidence of neurosyphilis among colored races may be more apparent than real. In support of this explanation are the following considerations: The hospitals and clinics reporting white patients with neurosyphilis are especially organized and equipped for discovering cases of this disease. A considerable proportion of these cases, one-half in some instances (Dennie and Smith,²⁰ Wile and

19. Fraser, A. R., and Duncan, A. G. B.: A Possible Explanation of the Increased Incidence and Early Onset of Neurosyphilis, *Brit. Jour. Dermat. & Syph.* **33**:251 (July) 1921.

20. Dennie, C. C., and Smith, D. O.: The Blood and Cerebrospinal Fluid in Three Hundred Known Cases of Syphilis, *Am. J. Syphilis* **2**:101 (Jan.) 1918.

Marshall²¹), which show abnormality of the spinal fluid, present no clinical signs of central nervous system involvement. Reports from China, on the other hand, have come from hospitals not so organized or equipped. Diagnoses have been made chiefly by men busy with surgery, on the basis of well marked physical signs alone. Pupillary abnormalities are more easily missed in dark-eyed races, and often the Chinese do not present the symptoms of nervous disorder which are typical for white patients. For example, I have found that the complaint of "lightning pains" among Chinese tabetics is rare.

The situation is illustrated in the case of the Peking Union Medical College hospital. The incidence of neurosyphilis among syphilitic patients in this hospital is about six times that reported by the fourteen other hospitals in China. Before the institution of a careful search for these cases, they had been seen no more frequently in Peking than elsewhere, but during a three year period in which a special syphilis clinic was maintained and free use made of the Wassermann laboratory and lumbar puncture needle, the number of cases among both inpatients and outpatients steadily increased. Thus, nineteen new patients with neurosyphilis were admitted to the medical ward during the first fourteen months and an equal number during the last four months of the period. During the whole three years, neurosyphilitic cases formed about 3.3 per cent. of the new medical admissions in Peking, against about 1.6 per cent. in the previously named hospitals in America.

With respect to outpatients of all departments, the percentage of cases of neurosyphilis for the three successive years was 0.11 per cent., 0.22 per cent. and 0.4 per cent., respectively. The average for the 35,000 patients seen during the whole period, 0.23 per cent., is the same as the average proportion reported for 68,000 patients from the three hospitals in America.

This rapid increase of cases was due in part to former patients and to physicians in other hospitals who referred cases for treatment. In larger part it was due to the well recognized relation between seeking and finding. Fewer cases in the clinic went unrecognized.

This fifth explanation probably will not account for all the observed racial differences in the incidence of neurosyphilis. Thus, Zimmermann²² found that neurosyphilis among patients in the same clinic was twice as common among whites as among blacks. It seems probable, however, that when hospitals in China have staffs and equip-

21. Wile, V. W., and Marshall, C. H.: A Study of the Spinal Fluid in One Thousand Six Hundred and Eighty-Nine Cases of Syphilis in All Stages, *Arch. Dermat. & Syph.* **3**:272 (March) 1921.

22. Zimmermann, E. L.: A Comparative Study of Syphilis in Whites and in Negroes, *Arch. Dermat. & Syph.* **4**:75 (July) 1921.

ment which will make thorough search possible, the present apparent differences in the nervous manifestations of syphilis in the white and yellow races will be markedly decreased.

DISTRIBUTION OF VARIOUS LESIONS.

The striking difference in the distribution of lesions is the high proportion of cord cases among the Chinese (Table 3).

Of the 26 per cent. with central nervous system lesions of vascular origin, six out of seven present a paraplegia due to transverse myelitis of the cord. Cases of general paresis, in contrast, are rare, possibly due in part to the fact that they present less urgent symptoms and less obvious physical signs.

ANALYSIS OF CASES

Since no series of cases of neurosyphilis in Chinese confirmed by spinal fluid examinations has been found in the literature,²³ it seems

TABLE 3.—INCIDENCE OF VARIOUS FORMS OF NEUROSYPHILIS AMONG HOSPITAL INPATIENTS IN CHINA AND AMERICA

General Hospitals	Number of Cases				Percentage of Cases			
	General Paresis	Tabes Dorsalis	Cerebro-spinal	Lesions of Vascular Origin	General Paresis	Tabes Dorsalis	Cerebro-spinal	Lesions of Vascular Origin
China.....	5	51	55	39	3	34	37	26
America....	481	299	223	—	48	39	22	—

worth while to record the principal findings in the group of sixty-five cases seen during the three year period in Peking. Some of these cases were under the charge of colleagues, Dr. Korns and Dr. Smyly. All the patients had definite physical signs of the condition, and in addition abnormality in the Wassermann reaction, cell count or globulin content of the spinal fluid. The series is too small to permit discussion of the interesting question of racial differences in symptoms and signs.

All the cases occurred in men as only men were admitted to the hospital. Twenty-six per cent. of these patients and 13 per cent. of all inpatients were soldiers. Soldiers were found particularly likely to have transverse myelitis, a fact which has been noted in other countries. All but six of the sixty-five gave a history of a penal sore, contracted on the average 5.4 years previously (10 years for patients with tabes and 3.3 years for patients with myelitis). For a few of the cases, the

23. Since this paper was written the following article has appeared: Woods, A. H.: Types of Cerebro-Spinal Syphilis in China, *China M. J.* **36**:206 (May) 1922.

Wassermann test was not available. Of the others, 75 per cent. gave a positive reaction in the blood serum and 65 per cent. in the spinal fluid. In 65 per cent., the spinal fluid globulin was increased; in all cases the cell count was above normal. The average count on the initial puncture was 87 cells per cubic millimeter.

The distribution of cases was: transverse myelitis 29, *tabes dorsalis* 16, cerebrospinal 14, hemiplegia 5, general paresis 1. The average initial cell count in the spinal fluid in these groups was: transverse myelitis 82, *tabes* 39, cerebrospinal 115, hemiplegia 73, general paresis 50. Besides the twenty-nine patients with transverse myelitis with positive evidence of syphilis, there were in the hospital during this period ten other patients with paraplegia not showing such evidence. Four fifths of the patients with myelitis due to syphilis had urinary retention or incontinence. Two thirds of the patients with cerebrospinal syphilis showed optic neuritis or atrophy. In one case there was a binasal hemianopsia. The frequency of these complications is perhaps due to the urgency of the symptoms.

Whatever is done for Chinese patients must be done quickly, or they go elsewhere. For this reason, and because of the comparatively small amount of the physician's time and patient's money required, intraspinal injections of mercurialized serum were given. Thirty-six of the patients received from one to nine such injections.

Too few of the patients remained under observation long enough to allow judgment concerning the value of this form of treatment. All cases showed prompt fall in spinal fluid cell count, but in only two did positive spinal fluid Wassermann tests become negative. Whether as a result of treatment or not, one or two of the apparently hopelessly paralyzed patients improved.

A soldier of 35, who had had a primary sore seven months previously, suddenly developed numbness of the legs and retention of urine. After eight days, he entered the hospital. At that time he had exaggerated knee reflexes with ankle clonus and a positive Babinski sign. Blood serum and spinal fluid both yielded four plus Wassermann reactions. The spinal fluid showed increased globulin and 900 cells per cubic millimeter. He was able to walk on admission, but complete paralysis of the legs came on rapidly, together with complete retention of urine. During 125 days in the hospital, he received seven intravenous injections of arsaminol and nine intraspinal injections of mercurialized serum. Successive spinal fluid counts were 900, 495, 90, 63, 30, 50, 20, 10, 5 and 3. Fluid Wassermann tests became negative, that of the blood serum remained two plus. Though increased knee reflexes and ankle clonus persisted, when he left the hospital he was able to walk and to urinate.

SUMMARY

Testimony of individual observers and reports of general hospitals agree that in China syphilis is relatively more common and neurosyphilis relatively less common than in America.

One explanation for this apparent racial discrimination is the probability that, for reasons named, many cases of neurosyphilis among the Chinese have been overlooked. In a teaching hospital in Peking, search for these cases revealed a proportion comparable with that found in teaching hospitals in America.

An unusual proportion of the Chinese patients have cord lesions of vascular origin.

Sixty-five cases of neurosyphilis are reported in which spinal fluid examination confirmed the clinical diagnosis.

A STUDY OF FOUR CASES OF GLOSSOPHARYNGEAL NEURALGIA *

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The anatomy of the glossopharyngeal nerve is a matter of dispute, and its exact relationship to the tenth and eleventh cranial nerves is matter for conjecture. All authorities differ with regard to the exact details of its central and peripheral connections.

According to Ranson,¹ the glossopharyngeal is a mixed nerve made up of general and special visceral afferent fibers and general and special visceral efferent fibers. The general and special afferent fibers have their cells of origin in the petrous ganglion. Their central branches terminate in the solitary tract and its nucleus. The general visceral efferent fibers arise from the cells of the inferior salivatory nucleus of the medulla oblongata. The special visceral afferent fibers arise in the nucleus ambiguus, the medullary continuation of the dorsolateral cell group of the anterior column of the cord.

Superficially the glossopharyngeal nerve takes its origin from the rostral end of the posterolateral sulcus of the medulla oblongata in line with the tenth and eleventh nerves. Like the vagus, the glossopharyngeal nerve has two ganglia. The dorsal root ganglion, the superior ganglion, is at the upper orifice of the jugular foramen; the trunk ganglion, the petrous ganglion, is in a small depression at the lower orifice of the jugular foramen. From the latter, branches of communication pass to the auricular branch of the vagus, to the jugular ganglion and to the sympathetic.

As the facial nerve emerges from the stylomastoid foramen it receives a branch of communication from the trunk of the ninth nerve, which passes through the posterior belly of the digastric. The first branch of the ninth nerve, according to Gray,² is the tympanic nerve, the nerve of Jacobsen, which forms the tympanic plexus from which arise the lesser superficial petrosal, a branch to the greater superficial petrosal, and branches to the tympanic cavity. The lesser superficial petrosal passes to the otic ganglion, a small, oval flattened mass below the foramen ovale on the medial aspect of the mandibular nerve, which

* From the Section on Neurology, Mayo Clinic.

* Read before the Minnesota Neurological Society, Rochester, Minn., May 13, 1922.

1. Ranson, S. W.: *The Anatomy of the Nervous System from the Standpoint of Development and Function*, Philadelphia, W. B. Saunders Company, 1921.

2. Gray, H.: *Anatomy of the Human Body*, Ed. 20, Philadelphia, Lea and Febiger, 1918, pp. 906-909 and 891-893.

surrounds the nerve to the internal pterygoid muscle. This ganglion receives fibers from the nerve to the internal pterygoid, from the seventh nerve, and from the sympathetic fibers accompanying the middle meningeal artery. The preganglionic fibers are derived from that part of the glossopharyngeal nerve which originates in the inferior salivatory nucleus. The postganglionic fibers pass through the auriculo-temporal nerve to the parotid gland.

The carotid branches of the ninth nerve communicate with the sympathetic on the carotid artery. The pharyngeal branches are composed of three or four filaments which unite posterior to the middle constrictor of the pharynx with the vagus and sympathetic to form the pharyngeal plexus. The muscular branch supplies the stylo-pharyngeus muscle. The tonsillar branches supply the palatine tonsil, the soft palate, and the fauces, and communicate with the palatine nerves. The lingual branches terminate in the papillae vallatae and the mucous membrane of the base of the tongue.

In 1918, Vernet,³ in discussing the syndrome of the jugular foramen, said that a destructive lesion of the ninth nerve causes paralysis of the superior constrictor of the pharynx which results in difficulty in swallowing solid food. Paralysis of the tenth nerve results in anesthesia of the soft palate and the posterior wall of the pharynx. Destruction of the accessory part of the eleventh nerve leads to paralysis of the soft palate and larynx, while destruction of the spinal portion produces paralysis of the sternocleidomastoid and trapezius muscles.

Trifacial neuralgia has long been accepted as a clinical entity. The characteristic paroxysmal pain, familiar to all neurologists and to most physicians, occurs in the area of distribution of one or more of the three divisions of the trigeminal nerve.

In 1907 and 1908, Hunt⁴ discussed in detail inflammations of a herpetic nature, involving the ear and adjacent regions. He attributed them to an involvement of the geniculate ganglion similar to involvement of the gasserian ganglion and dorsal root ganglions in herpes zoster. The pain was different from that typical of tic douloureux.

In 1908, Orbison⁵ reported a case of herpes of the tympanic membrane and endeavored to prove the condition "due to zosteroid affection of the petrosal ganglion."

3. Vernet, M.: Syndrome du trou déchiré postérieur (paralysie des nerfs) glosso paryngien, pneumogastrique (spinal), *Rev. neurol.* **34**:117-148, 1918.

4. Hunt, J. R.: On Herpetic Inflammations of the Geniculate Ganglion. A New Syndrome and Its Complications, *J. Nerv. & Ment. Dis.* **34**:73-94, 1907. A further Contribution to Herpetic Inflammations of the Geniculate Ganglion, *Am. J. M. Sc.* **136**:226, 1908.

5. Orbison, T. J.: Herpes of the Membrane Tympani: Due to Zosteroid Affection of the Petrosal Ganglion, *J. Nerv. & Ment. Dis.* **35**:500-506, 1908.

In 1910, Mills⁶ refuted Hunt's arguments concerning the nature of the afferent fibers of the facial nerve and attempted to show that the function of the nerve was chiefly gustatory and that only a vestigial remnant of common sensation remained over a small portion of the anterior part of the tongue and a narrow strip in the auricle.

Lesions of the glossopharyngeal nerve, experimental or acquired, are extremely rare. In 1896, Meyer⁷ described the changes in the taste buds of foliate type, of the tongue of the guinea-pig, subsequent to section of the ninth nerve.

In 1919, Neve⁸ described a case of what he termed herpes zoster of the glossopharyngeal nerve. The patient had peripheral facial nerve palsy on the left, impairment of the eighth nerve, vesicular eruption over the left half of the soft palate, and pain posterior and inferior to the left ear and down the left side of the neck.

Oppenheim⁹ described a case of paralysis of the ninth nerve in a woman. Objectively she had thermesthesia, paresis of the soft palate and pharyngeal muscles, and loss of taste in the soft palate, pharynx and posterior part of the tongue. Tactile sense was preserved intact.

In 1910, Weisenburg¹⁰ described a case of cerebellopontile tumor diagnosed for six years as tic douloureux. Frazier cut the sensory root of the fifth nerve and excised most of the gasserian ganglion relatively early, to relieve what appeared to be trifacial neuralgia. Following this operation the patient complained of pain in the right side of the throat radiating into the ear and neck and occasionally to the shoulder, and later he had severe pain at the root of the tongue and in the right side of the pharynx. This pain was paroxysmal and was set up by the contact of food or other foreign material with the mucous membrane of the pharynx. He also complained of pain in the lower right side of the face. It should be emphasized that the character of the pain differed after the ganglion operation in that before the pain was in the whole face as well as in the throat. Repeated pharyngeal and laryngeal examinations revealed excessive tenderness on the right side, but no weakness of any of the muscles of that region. At necropsy a tumor was discovered in the right cerebellopontile angle lying directly on the

6. Mills, C. K.: The Sensory Functions Attributed to the Seventh Nerve, *J. Nerv. & Ment. Dis.* **37**:273-284 and 354-377, 1910.

7. Meyer, S.: Durchschneidungs-versuche am Nervus Glosso-pharyngeus, *Arch. f. mikrosk. Anat.* **48**:143-145, 1896.

8. Neve, G. T.: Herpes Zoster of the Glossopharyngeal Nerve, *Brit. M. J.* **2**:630, 1919.

9. Oppenheim, H., quoted by Weisenburg; T. H.: *J. A. M. A.* **54**:1600 (May 14) 1910.

10. Weisenburg, T. H.: Cerebello-Pontile Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, *J. A. M. A.* **54**:1600-1604 (May 14) 1910.

sensory and motor roots of the fifth nerve. The ninth and tenth nerves, which were very prominent, stretched over the lower portion of the tumor. On account of the close relationship of the peripheral fibers of the fifth and ninth nerves, Weisenburg interpreted the pain which persisted in the area of distribution of the fifth nerve after resection of the major portion of the gasserian ganglion as a result of irritation of the ninth nerve. The pharyngeal pain was also attributed to irritation of the ninth nerve.

In 1920, Sicard and Robineau¹¹ described three cases of what they termed "algie vélo-pharyngée essentielle." The first two cases were seen in the French army in 1916 and 1917, respectively. In one case the pain was right sided; in the other, left. In neither case could a cause be established. There was no antecedent history of syphilis, and the condition did not respond to treatment for syphilis, to sedatives or physical agents. In both cases the pain had persisted over a period of several years; it was not continuous, but recurred frequently day and night in extremely acute paroxysms on the least movement of mastication, deglutition or speech. Occasionally the paroxysms occurred spontaneously. The patients developed suicidal tendencies on account of the apparent incurability of the condition.

The treatment advised consisted in section of the glossopharyngeal nerve and the pharyngeal branches of the vagus, with ablation of the superior cervical sympathetic ganglion. These two patients were operated on by surgeons unfamiliar with surgical procedures in the neck. In one the ninth nerve was cut, the sympathetic ganglion was removed, and the trunk of the vagus severed; in the other the ninth and twelfth nerves and the pharyngeal branches of the tenth nerve were cut, and the sympathetic ganglion was removed. Both of the patients convalesced rapidly. The first patient developed unilateral palsy of the vocal cord; the second, hemiatrophy of the tongue.

Sicard's third case was that of a woman, aged 50 years, in whom he successfully cut the ninth nerve and the pharyngeal branches of the tenth, and ablated the superior cervical sympathetic ganglion. During operation the trunk of the vagus was pulled, resulting in paresis of the homolateral vocal cord; this condition, however, was steadily improving. As in the first two instances, all pain disappeared immediately following operation. The first patient has been completely relieved for four years and the second for three.

Harris¹² has recently described two cases of a rare form of chronic paroxysmal neuralgia, which he termed glossopharyngeal neuralgia.

11. Sicard, R. and Robineau: *Algie vélo-pharyngée essentielle. Traitement chirurgical*, Rev. neurol. **36**:256-257, 1920.

12. Harris, W.: *Persistent Pain in Lesions of the Peripheral and Central Nervous System*, Brain **44**:557-571, 1921.

The pain, according to him, characteristically starts in the throat in the region of the tonsil and anterior pillar and radiates to the ear and upper part of the neck. The first case was that of a man, aged 40 years, whom he had first seen ten years before, when he successfully injected the third division on the affected side without influencing the pain. The second patient was a woman, aged 87 years, who had suffered from the condition for ten years. Harris was convinced of the involvement of the ninth nerve in these cases; he had seen a man with similar pains and intense hyperesthesia of the side of the neck, following a recurrence of epithelioma of the tonsil seven years after extirpation of a malignant vocal cord.

REPORT OF FOUR CASES OF GLOSSOPHARYNGEAL NEURALGIA OBSERVED
IN THE MAYO CLINIC BETWEEN MARCH 14 AND JUNE 1, 1922

CASE 1.—History.—Mr. W. H. L., a farmer, aged 63 years, came to the Clinic, March 14, 1922, complaining of pain in the throat and right ear. In 1912, he had had herpes zoster just above the left iliac crest. Five years before, after taking a drink of cold water he had had a sharp paroxysmal pain in the region of the right ear, associated with excessive tenderness of the right auricle. Similar seizures occurred sporadically until 1918, when his tonsils were removed. He was completely relieved for about three years, when, for six months, he had mild paroxysms. Following the extraction of several infected teeth in the fall of 1921, he was relieved until the last week in February, 1922, when the pain recurred while he was drinking water. At the time he came to the Clinic he had great difficulty in obtaining sufficient nourishment, owing to the pain induced by drinking and masticating. The pain was paroxysmal, sharp and agonizing; it arose in the right faucial region and radiated laterally to the area anterior and posterior to the right ear. It lasted from thirty to ninety seconds and was definitely increased in frequency by talking and chewing. At the onset he had attacks during the night; during the two weeks preceding his examination his distress had been chiefly diurnal.

Examination.—The physical examination was negative except for slight arteriosclerosis and moderate enlargement of the prostate. The systolic blood pressure was 118, the diastolic 80. Vision was 6/6 (with correction) in both eyes. Both pupils were slightly irregular. Reflexes and fields were normal. Both fundi were negative, except for slight choroidal sclerosis. A few peripheral lenticular opacities were noted. The Wassermann reaction was negative, as were the urinalysis, blood counts and stereoscopic roentgenograms of the head. On examination of the larynx and nasopharynx a trigger area was discovered deep in the hypopharyngeal region. The only neurologic findings were a slight to moderate diminution in perception of vibration and slight incoordination of both lower extremities.

Diagnosis: The neurologic diagnosis was trifacial neuralgia involving the auriculotemporal branch of the right mandibular division.

Treatment and Course.—On March 23, 1922, the sensory root of the right gasserian ganglion was cut, and the motor root preserved. The patient was greatly relieved. On April 8, there was complete anesthesia for tactile, pain and temperature senses over the area of distribution of the right trigeminal nerve. Sensation to touch and pain was normal in the external auditory canals and over the external aspects of both tympanic membranes. On April 14, the

patient returned to the Clinic. The pain had been recurring for the last forty-eight hours. During examination the paroxysms were almost continuous. Morphin, hypodermically, gave but little relief. On April 25, the glossopharyngeal nerve was evulsed and the pharyngeal branch of the vagus cut. For the last six weeks the patient has not had a recurrence of the pain. For a few days after operation he complained of a sense of fulness in the right side of the pharynx with a slight dysphagia. He has complete anesthesia to pain, temperature and touch over the area of cutaneous distribution of the

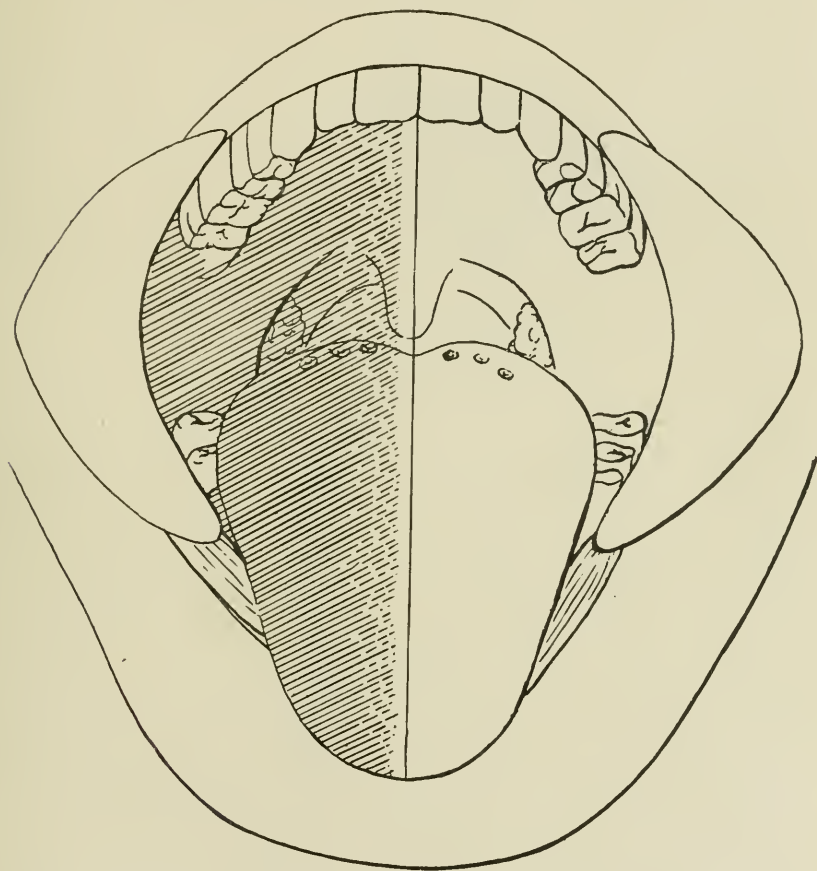


Fig. 1.—The shaded area shows the distribution of anesthesia to touch, pain and temperature over the tongue, hard and soft palates, uvula, tonsil and posterior pharyngeal wall on the right in Case 1, following the second operation.

right trigeminal nerve, over the mucous membrane of the right half of the buccal cavity, the right half of the alveolar processes, the tongue and hard palate and the anterior half of the right nasal cavity. Touch and pain are normally perceived over the entire surface of both external auditory canals, the external aspect of both tympanic membranes and both auricles. There is complete anesthesia to pain, temperature and tactile sensations over the entire right half of the inferior aspect of the soft palate. On its upper aspect the right

half of the soft palate is completely anesthetic for 1.5 cm. anterior to its posterior border. All of the portion of the right half of the pharynx lying between the level of the hard palate above and the pyriform fossa below is completely anesthetic to pain, temperature and tactile stimulation. Pain, temperature and touch are normally appreciated over the posterior half of the nasal cavity, and all of the nasopharynx above the level of a line drawn through the hard palate

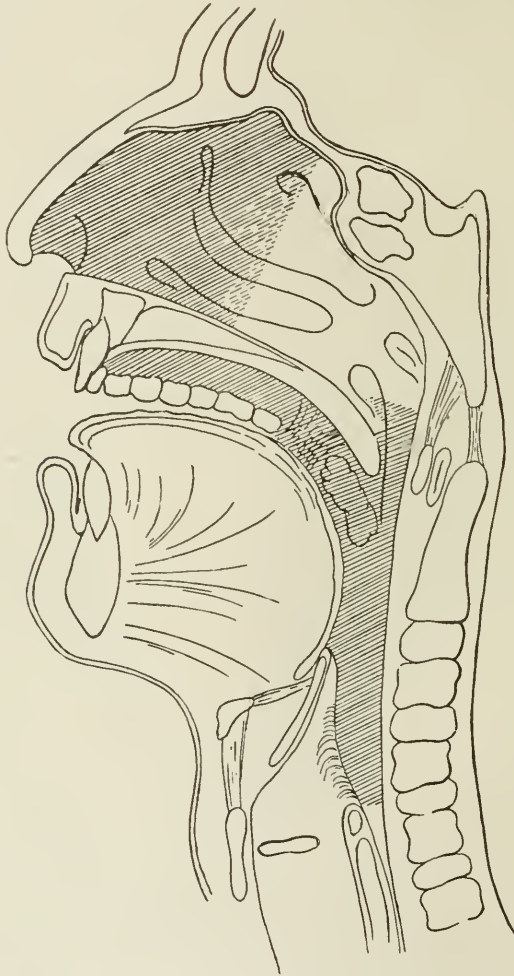


Fig. 2.—The shaded area shows distribution of anesthesia to touch, pain and temperature over the lateral wall of the nasal cavity, hard and soft palates and pharynx on the right in Case 1, following the second operation.

(Figs. 1 and 2). Sensation is normal over the epiglottis and the upper aspect of the larynx. Taste is absent for salt, sweet, bitter and sour over the entire right half of the tongue. There is paresis, Grade 2 to 3, of the right masseter and temporal muscles; the right pterygoid muscles are normal. The right

half of the soft palate, and the right superior, middle and inferior constrictors of the pharynx are totally paralyzed. There is loss of tone of about Grade 2, without actual loss of power, in the right half of the tongue.

CASE 2.—History.—Mr. F. W. McC., aged 52 years, came to the Clinic, March 28, 1922, complaining of a dull throbbing pain in the region immediately anterior to the right external auditory meatus, associated with paroxysms of sharp, stinging pain in the right side of the throat and right ear. He smoked a pipe incessantly. He had been deaf in the right ear for twenty years due to suppurative otitis media in childhood. He had never had herpes. Three weeks before his visit to the Clinic he had had a severe "cold" which lasted one week. As this infection was clearing up he noticed a sense of distress in the right side of the throat which he attributed to the cold. Two or three days later a dull throbbing pain developed in the region anterior to the right ear. The same day he began to have occasional, sudden, sharp, paroxysmal, stinging pains in the right side of the throat and right ear, lasting from twenty to forty seconds. By the second night he was beside himself, owing to the steady bombardment of paroxysmal pains. He was taking from eight to ten quarter grain (0.016 gm.) tablets of morphin daily. The narcotic relieved him of the dull throbbing pain; but mild paroxysms continued. After severe paroxysms he had a burning sensation and marked tenderness over the tragus. Talking and swallowing often induced attacks, although at times they occurred spontaneously.

Examination.—The patient was pasty and sallow. The systolic blood pressure was 90, the diastolic was 70, and the pulse was 58. Vision was 6/12 in the right eye and 6/6 in the left. The pupils were contracted (morphin?); the reflexes were impaired but present. The media were clear; the fundi were negative. There was a large perforation in the right tympanic membrane, but no evidence of active otitis media. The Wassermann reaction was negative, as were the urinalysis, blood counts and stereoscopic roentgenograms of the head. Neurologic examination was negative with the exception of diminution in hearing in the right ear.

Diagnosis: As in the first case, the diagnosis was trifacial neuralgia involving the auriculotemporal branch of the right mandibular division.

Treatment and Course.—March 31, 1922, the third division of the right trigeminal nerve was injected without influencing the pain. A day later the auriculotemporal nerve was injected, but the pain was not affected.

CASE 3.—History.—Mrs. E. M., aged 57 years, came to the Clinic, May 2, 1922, complaining of pain in the throat. With the exception of typhoid at 15 and pneumonia at 18, she had never been seriously ill. She had had ptosis of the right eyelid as long as she could remember. She had been perfectly well until the summer of 1921, when she developed occasional mild, sharp, paroxysmal pain, lasting a second or two, originating in the right side of the throat and radiating to the region around the angle of the right mandible. Gradually the paroxysms increased in severity, but they never came in series. After recurring several times daily for six or eight weeks the pain ceased as mysteriously as it had begun. On April 10, 1922, after she had taken a drink of cold water the trouble she had had during the summer of 1921 recurred suddenly. Now, however, the paroxysms continued in series over a period of three or four minutes. On April 17, she had a recurrence of paroxysms; since then they had recurred in attacks about twice a week. During all the attacks

in series she had felt that she was choking. She did not become cyanotic or really dyspneic. During the examination she had numerous paroxysms of pain. Although severe, the pain was not agonizing.

Examination.—The patient was a well preserved woman, moderately developed and fairly well nourished. Her blood pressure on two occasions was systolic 195, and diastolic 120; her pulse was 76. The blood Wassermann test was negative. The ear, nose and throat examinations were negative. Vision was 6/12 and 6/6 in the right and left eyes, respectively (with correction). The right pupil was slightly larger than the left; the reflexes were normal. Except for slight reduction in the caliber of the retinal arteries, the fundi were negative. Neurologic examination was negative, except for slight reduction in hearing in the left ear and ptosis, Grade 1 to 2, of the right upper eyelid.

Unfortunately, this patient left the Clinic before her examination was completed.

CASE 4.—*History.*—Mr. J. H., aged 61 years, came to the Clinic, April 10, 1922, complaining of pain in the region of the left external auditory meatus. As a child he had had "spotted fever" which resulted in the loss of the right eye. During adolescence he had had repeated attacks of suppurative otitis media; he had had a discharge from the left ear for ten years. From childhood until the age of 50, he had suffered from migraine. Nine years before he had become aware of repeated twinges of pain in the region immediately inferior and posterior to the left external auditory meatus. At first it was slight to moderate in severity; within thirty-six or forty-eight hours it became excruciating, and the attacks recurred at intervals of ten to thirty minutes. After eight to ten days the seizures diminished in frequency and by degrees ceased. Six years before he had a second attack lasting eight or ten days. Morphine alone gave relief. His third attack had occurred four years before and had lasted two or three weeks. The pain was not so severe or frequent as it had been. For a period of two weeks, commencing in the latter part of January, 1922, and ending in February, exceedingly severe attacks occurred at short intervals. His last attack commenced May 21, 1922, and was in progress at the time of examination. The pain was sharp, stabbing, and stinging; it came on suddenly in the area posterior and inferior to the left external auditory meatus, radiating down to the region of the angle of the left mandible. At onset the pain lasted only two or three seconds; in the fourth and last attack it persisted from thirty to fifty seconds. The pains never occurred in series of more than two or three paroxysms. Sudden movements of the head and neck, talking, drinking and fright seemed to induce paroxysms. Swallowing, especially of hot or cold fluids, was most likely to start the pain. A definite trigger area was not found.

Examination.—Physical examination revealed a man old beyond his years. He had a glass eye on the right, severe pyorrhea, systolic blood pressure 220 and diastolic 110, pulse 72. He had arteriosclerosis, Grade 3. The Wassermann reaction was normal. Vision of the left eye was 6/20 (with correction). The pupil was small and irregular; the reflexes were normal. The field was normal to rough test. The iris was atrophic; the globe was rather soft; and the pupil did not dilate. The fundus was seen with difficulty; there were no definite vascular changes. Ear, nose and throat examination revealed atrophic tonsils, a normal nasopharynx and larynx, a glazed appearance of the surface of the mucous membrane of the pharynx and slight irregularity of the nasal septum. When inspissated cerumen had been removed from the right external

auditory canal, the tympanic membrane was found thickened. The left external canal was filled in its internal two-thirds with fibrous and mucous polyps and mucopurulent exudate. Stereoscopic roentgenograms of the left mastoid showed cloudiness, Grade 3, while three biopsies on the polyps proved them to be myxomatous. The Bárány examination showed loss of function of the left vertical canal.

Treatment and Course.—As the patient was not a good operative risk the polyps were removed, and radical operation deferred.

Following the return of pain, May 21, the patient was seen in the neurologic section where the only additional findings on complete examination were static and movement tremors of moderate severity, and slight incoordination of the extremities. There was no loss of sensation to touch, pain, or temperature in the tongue, pharynx, hypopharynx, nasopharynx, soft palate, pillars, tonsils, and external auditory canals. Taste was preserved anteriorly and posteriorly over both sides of the tongue. During examination, May 24, he had a sudden attack of excruciatingly severe pain lasting about thirty seconds; it appeared and disappeared suddenly. During the paroxysm he coughed in a hacking manner every two or three seconds. Afterward he continued to hack, though less often, to relieve a sense of irritation in the left lateral pharyngeal region.

DISCUSSION

The distribution of pain in the first two cases was considered atypical, but the paroxysms were exactly like those of trifacial neuralgia. Following the recurrence of pain after section of the sensory root of the gasserian ganglion in the first patient, and after unsuccessful alcohol injections in the second patient, it became apparent that the condition was similar to trifacial neuralgia, but that some nerve other than the trigeminal was involved. Harris' discussion of glossopharyngeal neuralgia convinced me that the patients were suffering from that disturbance.

Two of the patients had hypertension (Cases 3 and 4) and one (Case 4) also had active disease of the middle ear. The second patient had no active disease, but had been deaf for twenty years as a result of suppurative otitis media in boyhood. The first patient had no such complicating factors, yet his paroxysms were more severe than those of the other three patients. No relationship could be established between these complications and the attacks of paroxysmal pain.

In the third and fourth patients, in addition to the paroxysms there were symptoms ordinarily interpreted as suggestive of irritation of the vagus. The third patient had a choking sensation with the attacks; the fourth coughed in a hacking manner throughout most of the paroxysms. In future studies attempts will be made to clarify this and other moot points. The possibility of the condition actually being vagoglossopharyngeal neuralgia must be considered.

The absence of taste following evulsion of the glossopharyngeal nerve and section of the pharyngeal branch of the tenth nerve brings

up the matter of the distribution of taste fibers. Luciani¹³ believes that the theory advanced by Carl,¹⁴ von Urbantschitsch,¹⁵ and first stated by Panizza¹⁶ in 1834, is the most acceptable. According to these authorities, taste fibers are derived entirely from the ninth nerve. The chorda tympani is composed of secretory and vasodilator fibers only. Unfortunately taste and ordinary sensation were not tested in the first patient prior to either of the operations. Hence, while the evidence is sug-

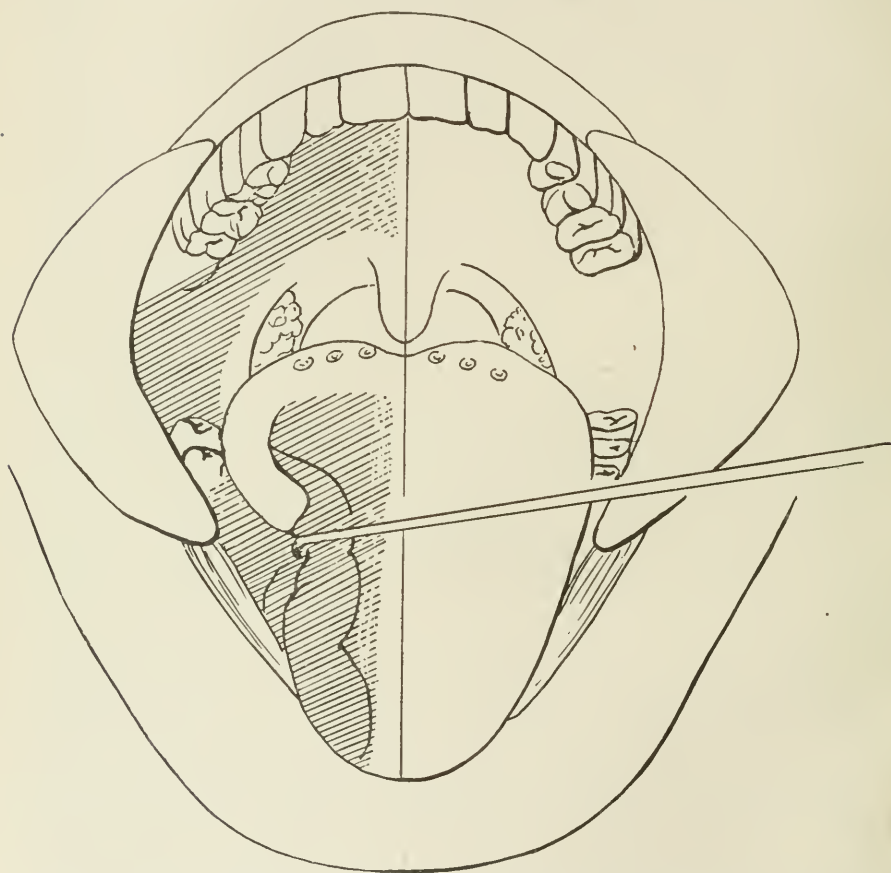


Fig. 3.—The shaded area shows the distribution of postoperative anesthesia to touch, pain and temperature over the tongue, hard palate and anterior part of the soft palate on the right, in a case of trifacial neuralgia.

gestive, it is not conclusive that the ninth nerve subserves taste to the tongue in its entire extent.

13. Luciani, L.: *Human Physiology*, translated by F. A. Welby, London, Macmillan Co. 3:401-405, 1915.

14. Carl, quoted by Luciani.

15. Von Urbantschitsch, quoted by Luciani.

16. Panizza, quoted by Luciani.

The preservation of sensation to tactile, pain and thermal stimulation in the posterior part of the right nasal cavity and the right half of the nasopharynx prompted an investigation of the sensory changes in the nasal, pharyngeal and buccal cavities in a patient in whom section of the sensory root of the gasserian ganglion for trifacial neuralgia had been performed.



Fig. 4.—The shaded area shows the distribution of postoperative anesthesia to touch, pain and temperature over the lateral wall of the nasal cavity, hard palate and anterior part of the soft palate on the right in a case of trifacial neuralgia.

REPORT OF CASE OF TRIFACIAL NEURALGIA

CASE 5.—Miss J. L., aged 42 years, came to the Clinic, May 2, 1922, complaining of paroxysmal, stabbing pain. The attacks lasted four or five months and had occurred every one or two years for seventeen years. The pain always started in the region of the mental foramen of the right mandible and darted posteriorly and upward.

On May 13, 1922, the posterior root of the gasserian ganglion was cut, the motor branch being preserved.

Examination revealed total anesthesia to tactile, pain, and thermal stimulation over the cutaneous distribution of the right trigeminal nerve. Pressure sensation over this area was about —3. The anesthetic fields in the nasal and buccal cavities are well illustrated in Figures 3 and 4.

According to Schafer and Symington,¹⁷ the sphenopalatine ganglion derives sensory fibers from the maxillary and glossopharyngeal nerves, and from the geniculate ganglion of the seventh nerve. Only a few of the fibers from the maxillary nerve enter the ganglion, most of them being distributed directly to the nose and palate. The nerve to the nasopharynx originates in the back of the ganglion, often in common with the Vidian nerve.

In consideration of these data it is clear that the nasopharynx and a variable portion of the posterior part of the nasal cavity receive their afferent fibers from a source other than the trigeminal nerve, and the trunk of the ninth. At the time of operation the glossopharyngeal nerve was evulsed from the jugular foramen. The actual rupture of the nerve occurred at the lower end of the petrous ganglion. For this reason it cannot be determined with any degree of certainty whether or not the tympanic branch of the ninth nerve was interrupted in its course. This leaves open the question whether the afferent fibers of the sphenopalatine ganglion concerned with the innervation of the nasopharynx are derived from the intermediate nerve through the geniculate ganglion, the greater superficial petrosal nerve and the nerve to the pterygoid canal, or from the small branch from the tympanic plexus to the greater superficial petrosal nerve.

On account of the nature of the operative procedure it is impossible to draw conclusions with regard to the respective parts played by the ninth and tenth nerves in the pharynx and adjacent regions.

The operation performed was not considered ideal because section of both nerves was made peripheral to their ganglions. In order to relieve the patient, however, the procedure advised by Sicard and Robineau was carried out in a modified form. Sufficient reason for the ablation of the superior cervical sympathetic ganglion was not presented. Very little is known about the pain due to irritation of the sympathetic ganglions. Pain of sympathetic origin is not well localized and is generally of a thermal quality. As yet sufficient time has not elapsed to warrant conclusions on the efficacy of the operation.

CONCLUSIONS

Glossopharyngeal neuralgia is a definite clinical entity, differing from trifacial neuralgia only in the area of distribution of pain.

The afferent fibers of the sphenopalatine ganglion concerned with the innervation of the nasopharynx are not derived from the trigeminal nerve.

17. Schafer, E. A., and Symington, J.: *Quain's Anatomy*, New York, Longmans, Greene & Co. 3:Pt. 2, 21-23, 1909.

LESIONS IN THE BRAIN OF A PATIENT WITH POSTENCEPHALITIC PARALYSIS AGITANS*

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MINNEAPOLIS

The termination of certain cases of epidemic encephalitis in a state similar to paralysis agitans has been the subject of common observation and much comment, but our knowledge of the pathology of this type has been largely deductive. That the acute cases have shown the most marked lesions in the basal ganglions, midbrain, pons and medulla, has been interpreted largely in the light of the work of Hunt¹ who described progressive atrophy of the globus pallidus in a case of the juvenile form of paralysis agitans. It has thus been supposed that the symptomatology of postencephalitic parkinsonianism is based on degenerations in the corpus striatum, but observations on the pathologic anatomy have largely been lacking. So far as I have been able to determine, the reports of Foix² and Goldstein³ are the only ones dealing with this question from the objective point of view. The following case is reported as a contribution to this subject.⁴

REPORT OF CASE

History.—A farmer, 51 years of age, admitted to St. Mary's Hospital, Minneapolis, June 30, 1922, complaining of lethargy, was perfectly well until two years before when he had epidemic encephalitis. He was confined to bed for about three months in a state of lethargy. He gradually improved so that after four months he was able to do a small amount of light work. He has, however, been continuously lethargic since the first and has often fallen asleep at unusual times and in peculiar situations, though not while working. During the last few weeks the lethargy has grown much more pronounced so that now he sleeps almost continuously. Vision has been poor since his acute attack.

The family history was negative. The patient was in robust health up to the time of the present trouble. He had certain childhood infections, from all of which he recovered without sequelae. There was no history of operations or injuries. He had never had headaches or any disease of the eyes, ears,

* From the Neuropathology Laboratory, University of Minnesota Medical School, Minneapolis.

1. Hunt, J. R.: Progressive Atrophy of the Globus Pallidus, *Brain* **40**: 58, 1917.

2. Foix, C.: Les lésions anatomiques de la maladie de Parkinson, *Rev. Neurol.* **28**:593, 1921.

3. Goldstein, K.: Ueber anatomische Veränderungen (Atrophie der Substantia nigra) bei postencephalitischen Parkinsonismus, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **76**:627 (May) 1922.

4. This case was studied clinically by Dr. C. C. Tyrrell of Minneapolis, and his clinical findings are presented in this report.

nose or throat; he had had no respiratory diseases and he did not have influenza at the time of the pandemic. He had had no cardiovascular, gastro-intestinal or genito-urinary diseases. Venereal infection was denied.

Examination.—July 1, 1922, examination revealed that the patient slept and snored continuously. He was easily awakened and then answered questions intelligently, but he did not converse spontaneously. His facial expression was fixed and masklike. The skin was rough and dry. The pupils were equal, small and irregular and did not react to light. The eyes were usually fixed straight ahead. There was no ptosis. A small amount of pus was present in the left conjunctival sac. The ears, nose and throat were normal. There was marked pyorrhea alveolaris; the tongue was dry and coated. The neck, lungs and heart were normal. Sclerosis of the peripheral vessels was moderate. There was some tenderness in the right upper and lower quadrants of the abdomen with a little rigidity, but no muscle spasm. No masses were palpable within the abdomen.



Fig. 1.—Fibrosis of the meninges over a sulcus in the region of the first left frontal gyrus. Hematoxylin and eosin; $\times 70$.

The upper extremities were continuously in a tremor indistinguishable from that of paralysis agitans. A "pill rolling" tremor of the fingers of the right hand was especially marked but was also present on the left side. The lower extremities were likewise tremulous and to such a degree that the constant rubbing had worn the skin and part of the flesh from the heels, leaving discharging sores. Spastic resistance was marked in all the skeletal muscles. The deep reflexes were all hyperactive but equal; there was no patellar or ankle clonus; the abdominal reflexes were active and equal; the plantar reflexes were normal.

The temperature (by axilla) was normal the first two days in the hospital, then varied between normal and 100 until the day of death, July 8, 1922, when

it rose to 103 F. The pulse ranged from 70 to 100 and the respiration from 20 to 25. The urine was normal. The blood examination showed a leukocytosis of 11,400. The blood Wassermann test was negative. No examination of the spinal fluid was made.

Clinical Diagnosis: Paralysis agitans following epidemic encephalitis.

Pathology.—Necropsy was performed eleven hours after death. Terminal bronchopneumonia, purulent bronchitis, chronic fibrous pleuritis and membranous adhesions about the ascending colon were found. There was no evidence of syphilis in the viscera or in the aorta.

The scalp and calvarium were normal. The dura was firmly attached to the pia-arachnoid by fine fibrous bands over the superior aspect of the brain, especially frontally and along the sides of the superior longitudinal sinus. On tearing the dura away, tags of these adhesions remained over the surface of the pia-arachnoid which were rather thick and opaque in some places, thin and transparent in others. The pacchionian bodies were distinct in their usual positions. The pia-arachnoid, especially over the frontal lobes, was thickened and



Fig. 2.—Camera lucida drawing of a cell in the left first frontal gyrus showing a slight amount of chromatolysis and four satellite cells. It is difficult to find many cells showing this amount of pathologic change in the cortex. Thionin; $\times 930$.

opaque, resembling at first glance a purulent exudate underlying a normal membrane. Gross section showed the thickening to be due to increase of fibrous connective tissue. It was especially marked along the vessels and over the sulci of the brain. There was some atrophy of the frontal lobes and a few adhesions on the under surface of the brain, especially marked anteriorly. The spinal cord was not removed.

The brain was put into 10 per cent liquor formaldehydi. Gross sections revealed no lesions visible to the naked eye.

Blocks of tissue were removed from various regions of the cortex, the corpus callosum, caudate and lenticular nuclei, thalamus, internal capsule, mid-brain, pons and medulla. The following strains were used: hematoxylin and eosin, thionin, polychrome methylene blue, Heidenhain's iron hematoxylin, Weigert's myelin sheath stain, Marchi's method for degenerating myelin sheaths

and Freeman's⁵ modification of the Warthin-Starry method for the demonstration of axis cylinders.

Sections from the cortex showed little abnormality except marked fibrous thickening of the pia-arachnoid, especially over the frontal lobes (Fig. 1). The thickening was due to simple fibrosis of the membrane without any evidence of an exudative inflammation. Tags of the torn adhesions which existed between the pia-arachnoid and the dura were seen microscopically as fibrotic projections from the superficial surface of the arachnoid. The cells, nerve fibers, interstitial tissues and blood vessels of the cortex were nearly normal throughout. There was some chromatolysis of the cortical neurons, not marked. Occasional cell bodies were found with three or four satellite glia cells closely applied to them (Fig. 2). There was no evident reduction in the number of cortical cells. Marchi and Weigert sections of the cortex showed normal myelin sheaths. No degenerating axis cylinders were demonstrable with the Freeman stain. The glia



Fig. 3.—Photomicrograph of a perivascular collection of small round cells in the left globus pallidus. Only a few vessels in this region are affected to this extent. Thionin; $\times 300$.

showed no evidence of proliferation, either of cells or of fibers. Occasional small vessels were seen surrounded by a few cells of the lymphocyte and plasma cell type. Thickening of the vessel tunics was no more marked than one would expect in a person 51 years of age.

The globus pallidus, putamen and caudate nucleus have been studied with especial care because of the prominence they have been given by Hunt¹ in the case of paralysis agitans. The results are disappointing from his point of view. These regions contained insignificant evidences of disease as compared with the lesions found in persons who have died in the acute stages of epidemic enceph-

5. Freeman, W.: A Silver Diffusion Method for Staining Nerve Fibers in Paraffin Sections, *Arch. Neurol. & Psychiat.* **7**:321 (March) 1922.

litis. With the exception of a moderate amount of chromatolysis and occasional examples of satellitosis, the cells of these regions were normal. There was no reduction in their number. The glia cells and fibers were not increased in the caudate nucleus or in the putamen. In the globus pallidus there was a slight increase in the number of glia cells in certain small areas, and a few cells resembling lymphocytes were present. Perivascular infiltration of small round cells was not observed in the putamen or in the caudate nucleus but was present, though by no means marked, in the globus pallidus (Fig. 3). The cells of the thalamus showed rather marked degenerative changes, largely in the form of chromatolysis, vacuolization of the cytoplasm, irregularity of the cell outlines and variations from normal in the position, size and shape of the nuclei. There was no evident reduction in the number of the thalamic neurons. Gliosis was more marked in this region than in the other parts of the basal ganglions, and perivascular collections of round cells were fairly frequent. Sections through

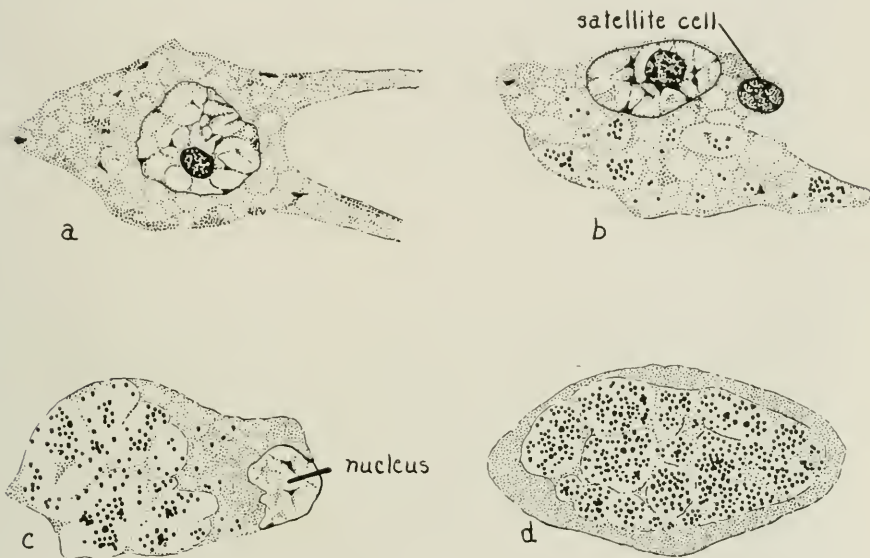


Fig. 4.—Types of degenerating cells in the nuclei of the oculomotor nerves. Compare with text. Camera lucida drawing; hematoxylin and eosin; $\times 840$.

the basal ganglions stained by the methods for studying axis cylinders and neurofibrillae and the myelin sheaths failed to reveal any degeneration of the nerve fibers. The internal capsules appeared normal. The ependymal lining of the third and lateral ventricles was normal.

Lesions were more pronounced in the mesencephalon than in any other situation. The nuclei of the oculomotor nerves were markedly affected. Practically all the cells of these nuclei were undergoing degeneration. The least injured were markedly chromatolytic with beginning vacuolization of the cytoplasm and irregularities in the shape of the nuclei (Fig. 4, *a*). From this type of cell every gradation could be traced through cells which were still more vacuolated with a few coarse yellowish pigment granules in the cytoplasm and in the vacuoles, and with more marked nuclear abnormalities (Fig. 4, *b* and *c*), to those cells which were only sacs containing considerable quantities of the



Fig. 5.—Degenerating myelin sheaths in the rootlet of the oculomotor nerve. Marchi's stain; $\times 80$.

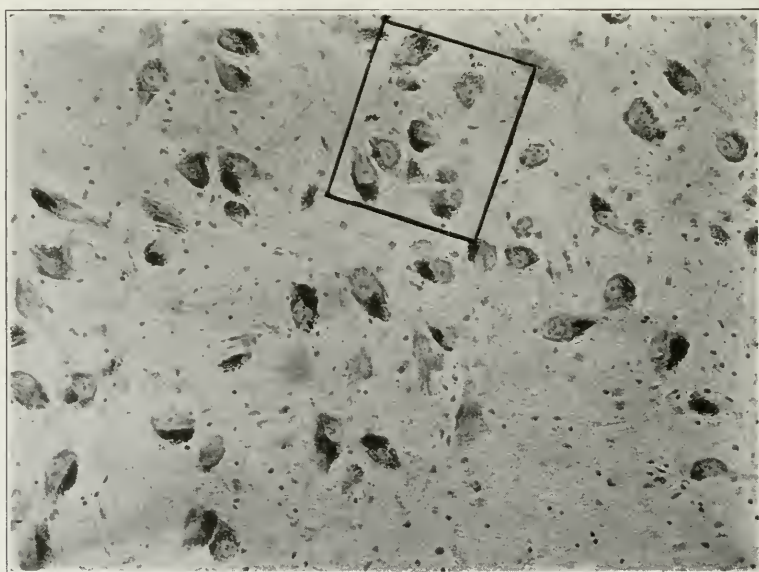


Fig. 6.—Photomicrograph of the cells in the substantia nigra of a woman 55 years of age, who died following a crushing injury of the thorax and pelvis in an automobile accident. Normal brain (N 345) for comparison. Polychrome methylene blue; 8 micron section; $\times 140$.

pigment granules (Fig. 4, *d*). Many of the cells had lost their processes altogether. Neuronophagia was not observed though an occasional satellite cell was seen (Fig. 4, *b*). The cells seemed merely to have undergone gradual disintegration without any chemotactic reaction. Parallel with the cell degeneration in the third nerve nuclei, sections of the third nerve rootlet stained with Marchi's, Weigert's and Freeman's stains all showed degeneration of a marked degree (Fig. 5). Increase in the number of glia cells was pronounced. Large clear glia nuclei were common and numerous elongated cells (Stäbchenzellen) were seen. Glia fiber proliferation was not marked. Perivascular infiltration of small round cells was common to most of the vessels, large and small, though the amount of infiltration was insignificant when compared with that in the acute cases. No abnormalities were observed in the colliculi. There were no marked lesions of the nucleus ruber. The ependyma of the aqueduct appeared normal.

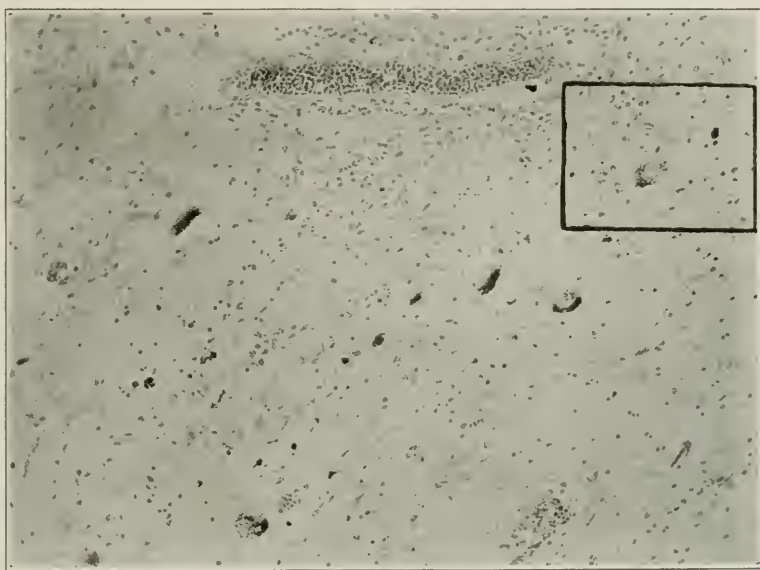


Fig. 7.—Photomicrograph of the substantia nigra in the case of post-encephalitic paralysis agitans. The loss of substantia nigra cells, the perivascular collections of round cells, the perivascular and diffuse gliosis, and the scattered pigment from broken-down cells are all evident when compared with the normal case (Fig. 6). Polychrome methylene blue; 8 micron section; $\times 140$.

The substantia nigra of either side was more profoundly altered than any other cell group. The cells were reduced to a small fraction of the normal number (Figs. 6 and 7). Those remaining were without exception chromatolytic; at least, this was the case in those cells in which the pigmentation was not so pronounced as to obscure the structure of the cytoplasm. No cells could be found as large as those of average size in normal brains used for comparison. The pigment in the cells appeared to be increased, but this was probably only relative, due to the shrinkage in the size of the cells which had crowded the pigment granules more closely together. Some of the cells contained little

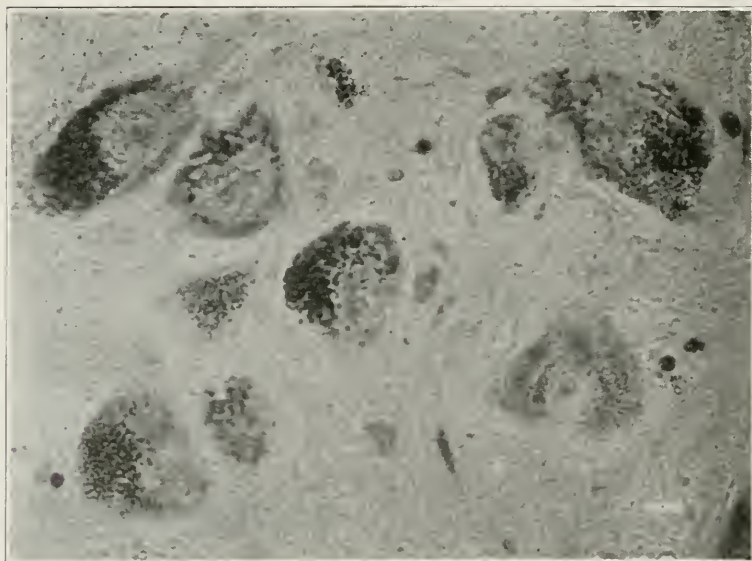


Fig. 8.—Photomicrograph of the field outlined in Figure 6. Normal substantia nigra cells for comparison with Figure 9; $\times 570$.



Fig. 9.—Photomicrograph of the field outlined in Figure 7. One of the surviving substantia nigra cells is shown. The scattered pigment from the previously degenerated cells and the gliosis are evident; $\times 570$.

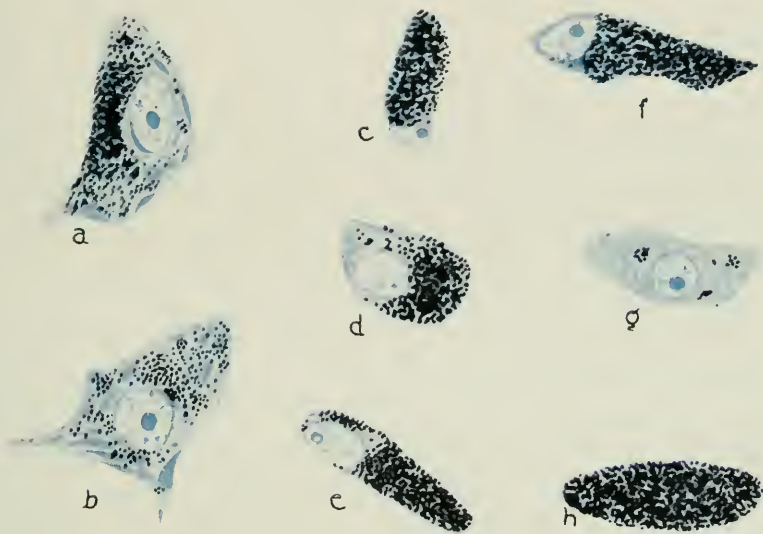


Fig. 10.—Camera lucida drawing of substantia nigra cells. Cells *a* and *b* are taken from the normal case (N 345). Cells *c* to *h* are from the case of post-encephalitic paralysis agitans. Atrophy of the cells, chromatolysis, nuclear abnormalities and relative increase in the intracellular pigment are evident in the postencephalitic case. The cells are all reproduced on the same scale of magnification from 8 micron sections stained with polychrome methylene blue; $\times 600$.

pigment; such cells were also found in normal cases. Decrease in size of the nuclei with marked eccentricity of the nuclear position was a regular finding. Neuronophagia did not occur in connection with these cells, and even examples of satellitosis were hard to find. Sections stained to demonstrate axis cylinders showed marked decrease in number of fibers; many of the remaining fibers were curled in the form of spirals and were irregularly swollen. That many cells had disappeared was evident, not only from their absence and from the absence of their processes, but also from the fact that within the boundaries of the substantia nigra there was more pigment scattered free in the interstitial network and in the glia cells than there was in the remaining nerve cells. This pigment was also found piled up along the walls of many of the vessels of the region, apparently in the process of being carried away along the perivascular spaces by migratory large mononuclear cells which had ingested

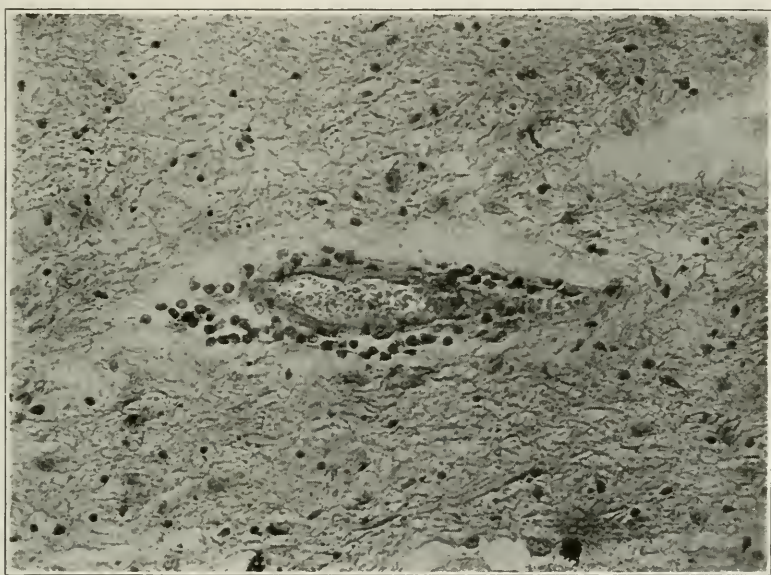


Fig. 11.—Perivascular infiltration of small round cells in the substantia nigra. A moderate spongy gliosis around the vessel and diffuse increase of glia cells are present. Hematoxylin and eosin; photomicrograph: $\times 240$.

it. Increase of glia cells was marked, but glia fiber proliferation did not appear to have kept pace with the cell increase. The gliosis followed closely the position of the substantia nigra, except for the region of the third nerve nuclei, as already noted. Perivascular infiltration of round cells (lymphocytes, plasma cells and large mononuclears, many of the latter containing an abundance of pigment granules) was more marked in this region than elsewhere. There were numerous fibroblasts scattered among the perivascular round cells; this is evidence of chronicity with a tendency toward healing which has not been noted in patients dying in the acute stages of epidemic encephalitis. The vessels were not any more sclerotic, however, than would be expected in a person of the patient's age.

The changes in the substantia nigra described in the foregoing are pictured fairly satisfactorily in the accompanying photomicrographs and drawings (Figs. 6-11); the captions are sufficiently descriptive to make it unnecessary to consider at this point the individual lesions illustrated.

Sections of the pons, medulla and the first cervical segment of the cord showed lesions of minimal degree. Chromatolysis was noted in many of the cells, but other cellular abnormalities were lacking. The occasional small areas of spongy gliosis may be interpreted as the end-results of the previous inflammation which undoubtedly involved this region during the acute stage of the disease. Perivascular collections of round cells occurred here, but they were far less extensive than in the midbrain. Some of the vessels were moderately sclerotic. The ependyma of these regions appeared normal. There were no lesions in sections from the cerebellum.

Weigert sections showed the same kind of thinning out of the myelin sheaths in patchy areas, especially in the midbrain and in the corpus callosum, as Spiller⁶ has recently described in the pons, medulla and cord in his case

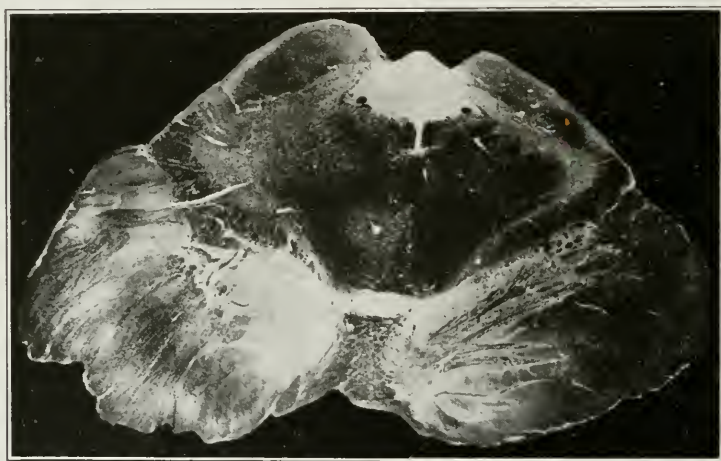


Fig. 12.—Photomicrograph of a Weigert preparation of the midbrain showing patchy areas in which the myelin sheaths are absent. The loss is especially marked in the peduncles; $\times 3$.

(Fig. 12). Corresponding degeneration of the axis cylinders does not appear to have occurred (except in the region of the substantia nigra, as already noted), so that the patches in this case reminded one very much of those seen in multiple sclerosis.

SUMMARY AND COMMENT

The patient had an attack of epidemic encephalitis with gradual recovery. He then developed the symptoms of paralysis agitans and entered on a stage of chronicity during which he became lethargic and

6. Spiller, W. G.: Epidemic Encephalitis with Myelitis, *Arch. Neurol. & Psychiat.* **7**:739 (June) 1922.

finally died. The pathologic findings are in accord with such a progress of events, and confirm the opinion of von Economo,⁷ Globus and Strauss,⁸ and Schaller and Oliver,⁹ that epidemic encephalitis may become chronic with a lethal termination.

The principal interest in this case lies in the degeneration of the substantia nigra without extensive involvement of the lenticular nuclei, considering that the patient presented the typical clinical picture of Parkinson's disease. The case does not stand alone in this. Goldstein³ has described and pictured in a number of cases (he does not state how many) of postencephalitic parkinsonianism, lesions in the substantia nigra which are similar in every important respect to those here presented. Foix² has reported a similar case. He found:

"1. Massive lesions of the substantia nigra, discrete lesions of the lenticular nucleus, the optic thalamus, the tegmentum pontis and the colliculi."

"2. Persistence of fully active inflammatory lesions."

Foix has also studied the pathology in seven cases of classical paralysis agitans. In all of these he found abnormalities of the substantia nigra and of the lenticular nuclei, but the changes in the substantia nigra were constantly more intense than those of the lenticular region.

Souques and Trétiakoff¹⁰ describe the pathologic changes in the brains of three cases of paralysis agitans. In all of them degeneration of the substantia nigra was found and no appreciable lesions of the globus pallidus could be determined.

Souques¹¹ refers to some of Trétiakoff's work, the original report of which I cannot find, in which the latter studied nine cases of bilateral paralysis agitans and one unilateral case. In the nine bilateral cases he found bilateral lesions of the substantia nigra and in the unilateral case degeneration of the substantia nigra only on the side opposite the symptoms.

A fairly complete exposition of opposing opinions as to the pathology of paralysis agitans and the physiology of the basal ganglions and

7. Von Economo, C.: Ein Fall von chronischer schubweise verlaufender Encephalitis lethargica, München. med. Wchnschr. **66**:1311 (Nov. 14) 1919.

8. Globus, J. H., and Strauss, I.: Subacute Epidemic (Lethargic) Encephalitis, Arch. Neurol. & Psychiat. **8**:122 (Aug.) 1922.

9. Schaller, W. F., and Oliver, J.: Chronic Epidemic Encephalitis. Report of a Case: Clinical Record, Complete Necropsy and Detailed Histologic Study of the Central Nervous System, Arch. Neurol. & Psychiat. **8**:1 (July) 1922.

10. Souques, A., and Trétiakoff: Lésions du locus niger dans trois cas de paralysie agitante, Bull. et mém. Soc. méd. d. hôp. de Paris **36**:1027, 1920; abstr., Rev. Neurol. **28**:773, 1921.

11. Souques, A.: Rapport sur les syndromes parkinsoniens, Rev. neurol. **28**:534, 1921.

extrapyramidal motor system is presented in Souques' ¹¹ paper and in the discussion which follows it. Probably the cases with the paralysis agitans syndrome following epidemic encephalitis offer a better opportunity for settling the vexatious questions surrounding paralysis agitans and the functions of the various nuclei involved than the cases of paralysis agitans proper. At present it seems likely that lesions of the substantia nigra in relation to the parkinsonian syndrome will become more and more significant.

THE PATHOLOGIC FINDINGS IN THE HEART MUSCLE IN PROGRESSIVE MUSCULAR DYSTROPHY*

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NEW YORK

While observing the clinical course of several cases of progressive muscular dystrophy, my attention was called to the manner in which patients who had remained in statu quo for fairly long periods of time, would, without warning, suddenly develop in rapid succession hypostatic pneumonia, pulmonary edema, hydropericardium and hydrothorax, culminating in death. Cardiovascular disturbance seemed best to explain these phenomena, and in two cases reported by Goodhart and myself,¹ lesions were found in the heart muscle, essentially of the same character as those found in the skeletal muscles.

In remarkably few cases of pseudohypertrophic muscular atrophy has a study of the heart muscle been made. Oppenheim,² referring to the few cases recorded,³ without giving reason for his opinion, says that he considers myopathic involvement of the heart muscle doubtful. Marinesco,³ in his splendid monograph on the disease of muscles, devotes a few paragraphs to the changes in the heart muscles occurring in the course of progressive muscular dystrophy. He says: "Among the muscles which offer an extraordinary resistance to the invasion by the pseudohypertrophic myopathy we must consider in the first place the cardiac muscle It is also true," he adds, "that publications in regard to this question are extremely rare, but this, perhaps, can be explained best by the fact that few investigators give sufficient attention to the microscopic examination of this organ. Nevertheless, Coste

*From the Department of Pathology of the Mount Sinai Hospital, New York City. This work was carried out during the tenure of the Sachs Fellowship in Neuropathology.

* Read before the New York Neurological Society, April, 1922.

1. Goodhart, S. P., and Globus, J. H.: On the Nature of Muscular Dystrophies with a Report on the Changes in Cardiac Muscle in Two Cases. *Neurol. Bull.* 1:387, 1918.

2. Oppenheim: *Textbook of Nervous Diseases* (translation by Alexander Bruce), Chicago, Chicago Medical Book Co., Ed. 5, 1911, p. 246.

3. Ross, James: On a Case of Pseudo-Hypertrophic Paralysis, *Brit. M. J.* 1:200 (Feb. 3) 1883. Hammond: Cited by Oppenheim and Marinesco. (Original article not available.) Stembo: Nimmt das Herz an der Pseudo-hypertrophie in den Fällen von Muskel-pseudo-hypertrophie? (Demonstration before Medical Association Vilna Nov. 12, 1897.) Cited by Oppenheim and Marinesco. (Original article not available.) Marinesco, G.: *Maladies des Muscles*, *Nouv. traite de méd. et de thérapeutique*, 1910, pp. 34 and 110.

and Cioja,⁴ Rinicker,⁵ Goetz⁶ and Hammond³ have observed hypertrophy of the heart in pseudohypertrophic paralysis without determining whether it was inherent in the disease or a coincidental complication. Mme. Sacara⁷ has noted, in four cases, irregularities in rhythm and intensity of the heart beat, while Jacobowitz has noted marked retardation of the pulse in one case. Stembo,³ employing the radio-

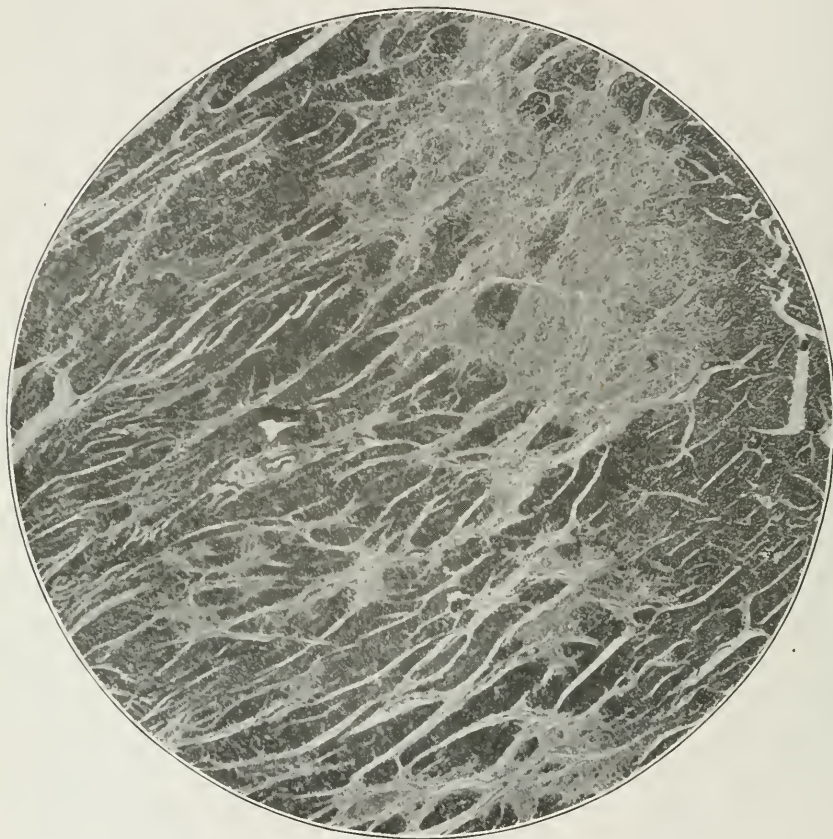


Fig. 1.—Section of the wall of the left ventricle showing a large typical fibrous scar with fairly conspicuous bundles of connective tissue radiating from it.

4. Original article not available.

5. Rinicker: *Verhandlungen der Physikalisch-medizinischen Gesellschaft in Würzburg*, 1860, p. 15. Rinicker's case of progressive muscular dystrophy, in a boy of 8½ years of age, showed enlargement of the heart during life. No anatomic findings are reported.

6. Goetz: *Beitrag zur Atrophie Muscularum Lipomatosa*, *Aertz. Intelligenz Bl.*, München 1879, p. 419.

7. Sacara-Tulbove: *Contribution à l'étude clinique de la paralysie pseudo-hypertrophique*, *Rev. méd.* **14**: 1894.

scope, was convinced that the heart in his case was affected by the disease, as it showed evidence of hypertrophy. Laignel-Lavastine⁸ described cardiac palpitation without valvular lesion as a rare complication of a case of primary myopathy of the Landouzy-Déjerine type.

In the chapter on the pathologic anatomy of muscular dystrophy, Marinesco³ describes a case in which the heart appeared involved and

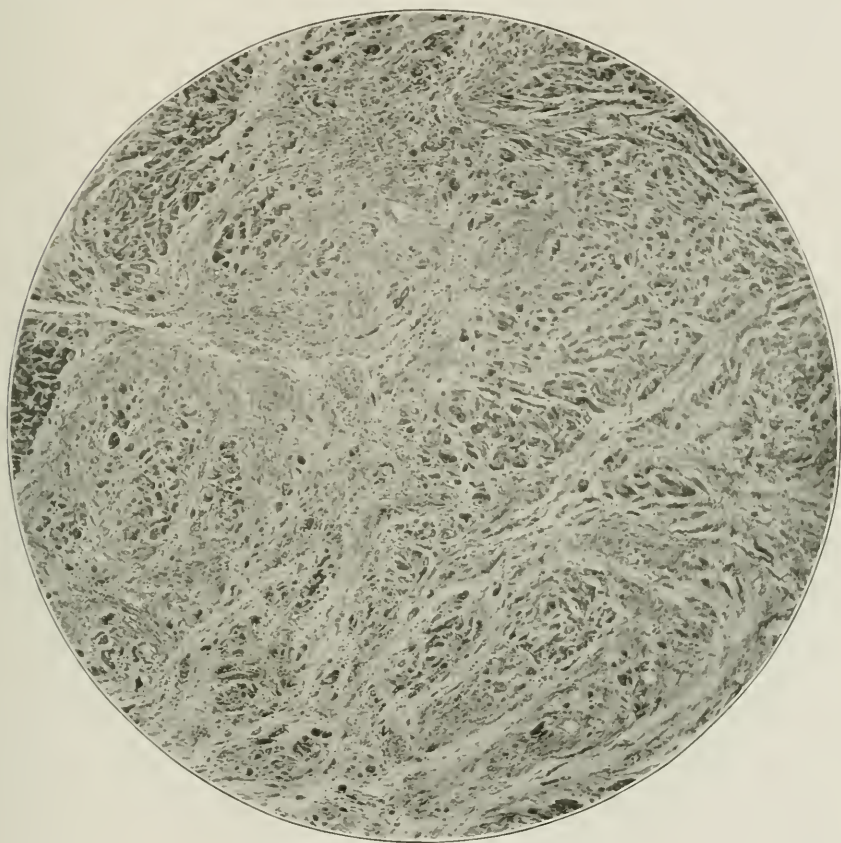


Fig. 2.—Section through a large scar in the wall of the left ventricle showing a marked increase in the interstitial connective tissue with a few small bundles of muscle fibers isolated in the scar.

showed typical lesions. He says, "In the study of the symptomatology of the primary myopathies one notes that the cardiac troubles do not generally participate in the general ensemble of characteristic phenomena of this disease, though the occurrence of myocardial disturbances has already been indicated." He adds that in the case of a child who died

8. Laignel-Lavastine: Myopathie primitive progressive, *Arch. gen. de méd.*, n. s. **5**:185, 1901.

of pulmonary tuberculosis complicating pseudomuscular hypertrophy, he found definite lesions in the cardiac muscle fibers and multiplication of their nuclei with increase of the interstitial tissue. Thus it appears that Marinesco inclines toward the belief that the cardiac muscle, in spite of its peculiar resistance to the disease, may be involved in the myopathic process.

Eulenberg and Cohnheim⁹ reported the heart in one case as normal in size and well contracted but of a rose-violet color. Charcot¹⁰ found

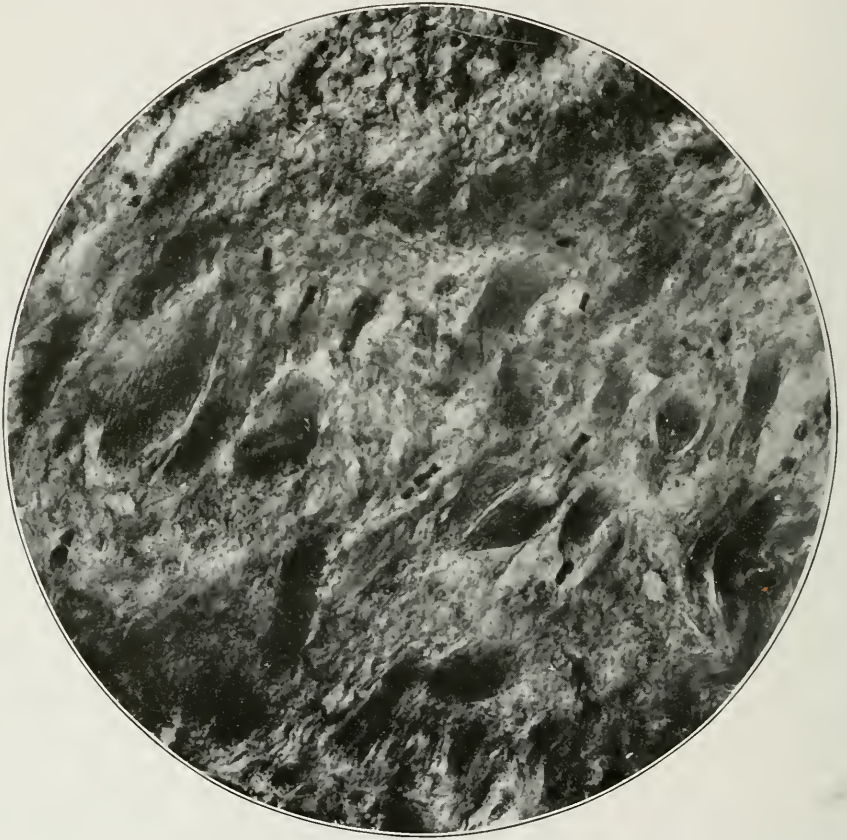


Fig. 3.—Section showing fragmentation of cardiac muscle fibers and marked proliferation of the interstitial connective tissue.

the muscle of the left ventricle free from pathologic change. In one case, Middleton¹¹ found the heart to be rather small and pale, but

9. Eulenberg and Cohnheim: *Verhandlungen der Berliner Mediz. Gesellsch.* **1**:191, 1865-1866. Cited by Potter. (Original article not available.)

10. Charcot: *Arch. de physiol.* **10**:228, 1871-1872.

11. Middleton, G. S.: *On the Pathology of Pseudo-Hypertrophic Muscular Paralysis with Remarks on a So-Called Degeneration of the Nervous System*, Glasgow M. J. **22**:81, 1884.

there was no marked increase in fat deposit and no hyaline degeneration. There was moderate increase of interstitial connective tissue. Goetz¹⁰ reported enlargement and paleness of the heart and the occurrence of yellowish patches in the papillary muscle about 1 cm. deep.

Ross³ reported definite myocardial involvement in a boy 12 years of age, who suddenly developed diarrhea, vomiting and general prostration culminating in sudden death. The heart muscle was flabby, the ventricles distended, the right ventricle filled with blood, the left

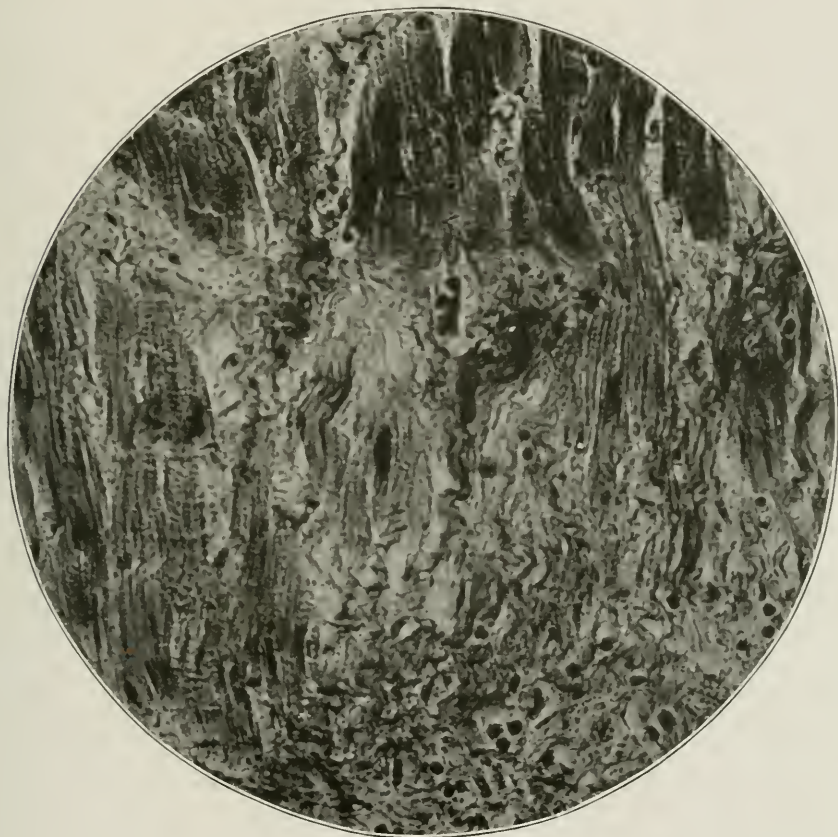


Fig. 4.—Section through a papillary muscle in the wall of the left ventricle showing interruption in the course of the muscle fibers by advancing scars.

empty. The wall of the left ventricle was one half inch (1.27 cm.) thick; the muscle was pale yellow and easily torn. The auricular walls were covered with a layer of fat. The heart muscle showed many atrophied fibers, which in many places were separated by interstitial connective tissue, increased in amount. Cardarelli,¹² describ-

12. Cardarelli: *Policlinico* 3:453, 1896 (Med. Section).

ing the heart of a boy 15 years of age, mentions only abnormal increase in the subepicardial fat. In his case, a severe attack of diarrhea and prostration led to sudden decline and death. Sachs and Brooks¹³ conclude that in their case the cardiac involvement formed no part of the myopathy. While the myocardium showed "diffuse fibroid infiltration," "the muscle fibers were found everywhere in a very natural condition," with neither atrophy nor abnormal pigmentation. Bunting,¹⁴ in one

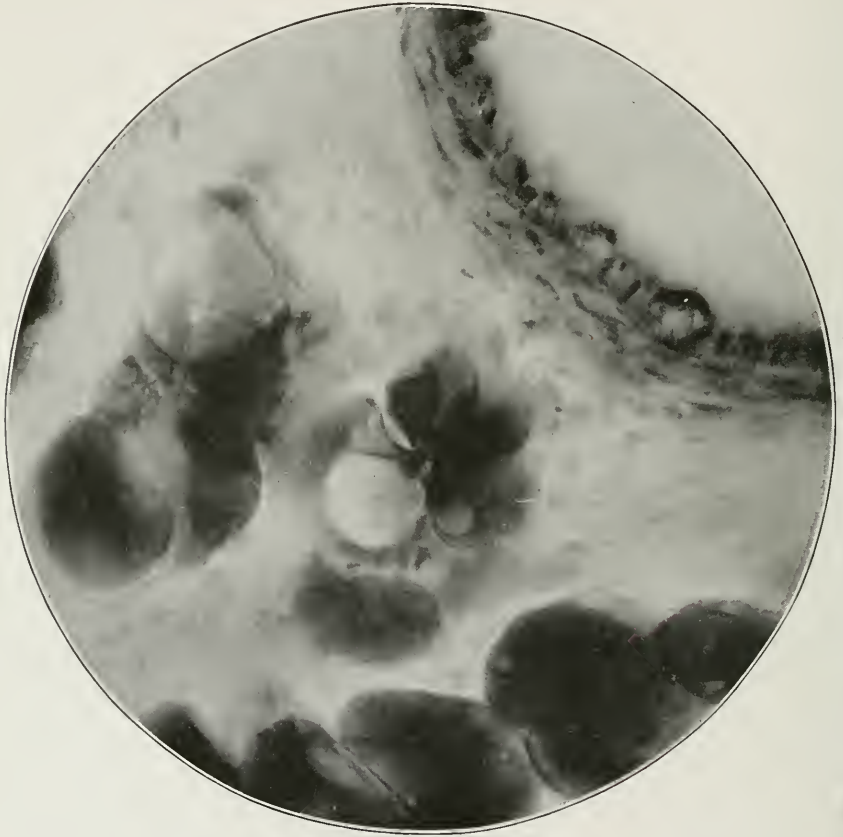


Fig. 5.—Section of left ventricle showing a vessel with the adventitia infiltrated with large masses of fat.

case, gave particular attention to involuntary muscle, striated (cardiac) and smooth (stomach). The heart was small and weighed 100 gm.; the valves were normal; the muscle was pale—grayish-pink. Sections

13. Sachs and Brooks: *Am. J. Med. Sc.* **122**:54, 1901.

14. Bunting, C. H.: *Chronic Fibrous Myocarditis in Progressive Muscular Dystrophy*, *Am. J. Med. Sc.* **125**:244, 1908.

of the wall of the left ventricle showed many translucent patches, irregularly distributed, which were most often found directly beneath the epicardium, with the subepicardial fat invading the muscle. The vessels were free from sclerotic changes. The lesions in the heart were identical with lesions in the skeletal muscle. He also reported increase in fibrous connective tissue and atrophy of muscle fibers in the wall of the stomach. More recently, Potter¹⁵ reported a case of muscular

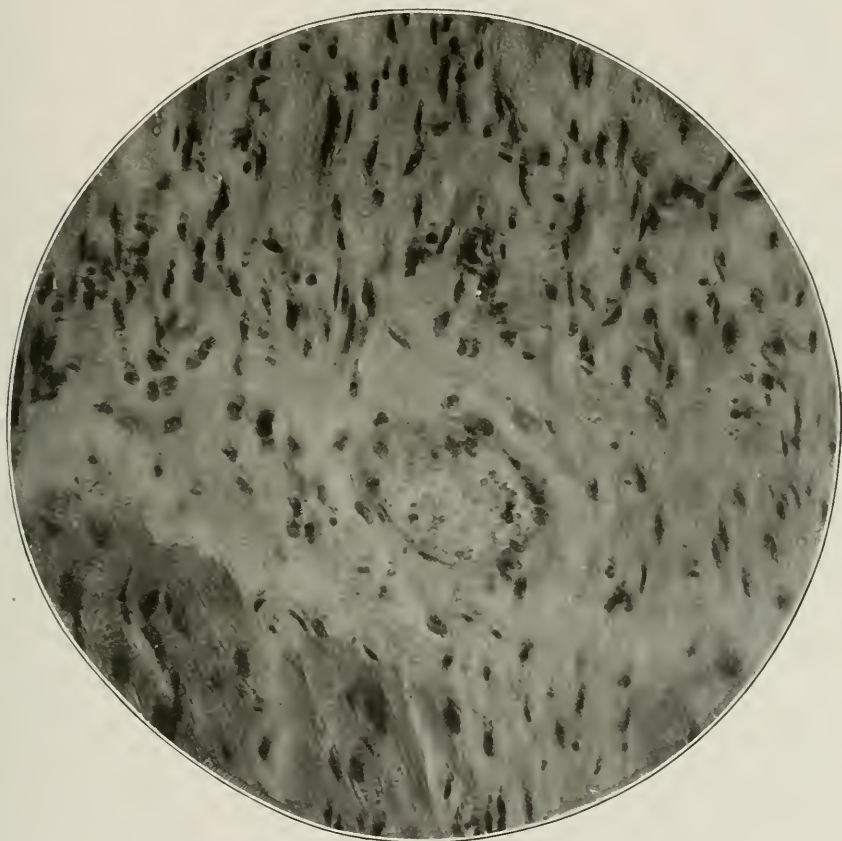


Fig. 6.—Section through the wall of the left ventricle showing a typical scar surrounded by a zone in which the muscle fibers show features typical of the skeletal muscles. There is also a distinct accumulation of fixed connective tissue cells about the blood vessel.

dystrophy with definite changes in the cardiac muscle, which he believed constituted a part of the myopathic disease.

15. Potter, F. C.: A Pathologic Study of a Case of Pseudo-Muscular Dystrophy with Change in the Heart Muscle. *New York M. J.*, Aug. 28, 1909.

REPORT OF A CASE

Clinical History.—A boy, 5 years of age, was examined by Dr. Roberto in 1916. His gait was unsteady. Calves and deltoids were well developed, but the pectoral muscles showed marked wasting. He had great difficulty in rising from the sitting posture and on rising from a recumbent posture had to climb up on his legs. A diagnosis of progressive muscular dystrophy was made. He gradually grew weaker and in June, 1919, it was noted that while there was no evidence of an infection, the pulse was rapid and variable, ranging from

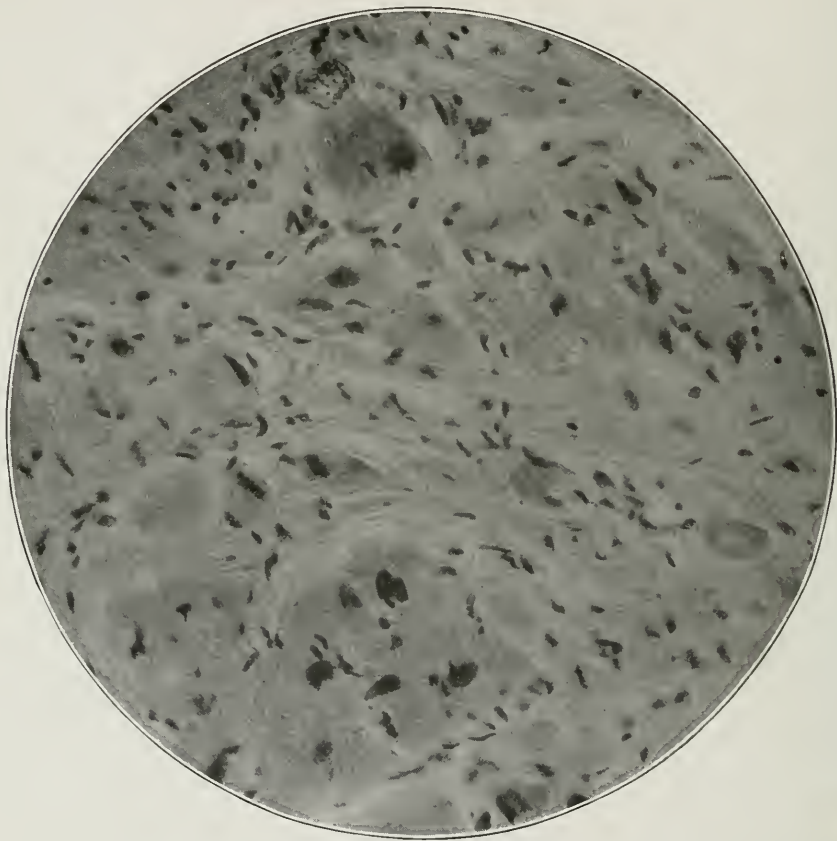


Fig. 7.—Section through the wall of the left ventricle showing fragmentation and multiplication of nuclei and infiltration of the interstitial tissue by fibroblasts.

80 to 130. Frequent changes occurred without apparent cause. The arms were so weak that he had to be fed. In October, 1921, he was still weaker. Following the extraction of a tooth, there was collapse and rapid pulse. A large amount of acetone was found in the urine. There was continual vomiting, followed by a convulsion, and he died twenty-four hours after the operation.

Necropsy Findings.—The pectoralis major and minor, the deltoid, the intercostals, the diaphragm, the rectus abdominis and the obliquus externus and

internus (the muscles examined) showed advanced changes of progressive muscular dystrophy. The lungs were voluminous, quite edematous, the upper lobes fairly well aerated but the lower lobes heavy and hyperemic, with several small pneumonic areas. The heart was somewhat smaller than normal, the left ventricle well contracted and empty, the right dilated and filled with blood. The subepicardial fat was somewhat increased, and some fatty streakings were found under the endocardium. The heart muscle was pale, yellowish-red, flabby and friable. On section, the wall of the left ventricle showed many translucent

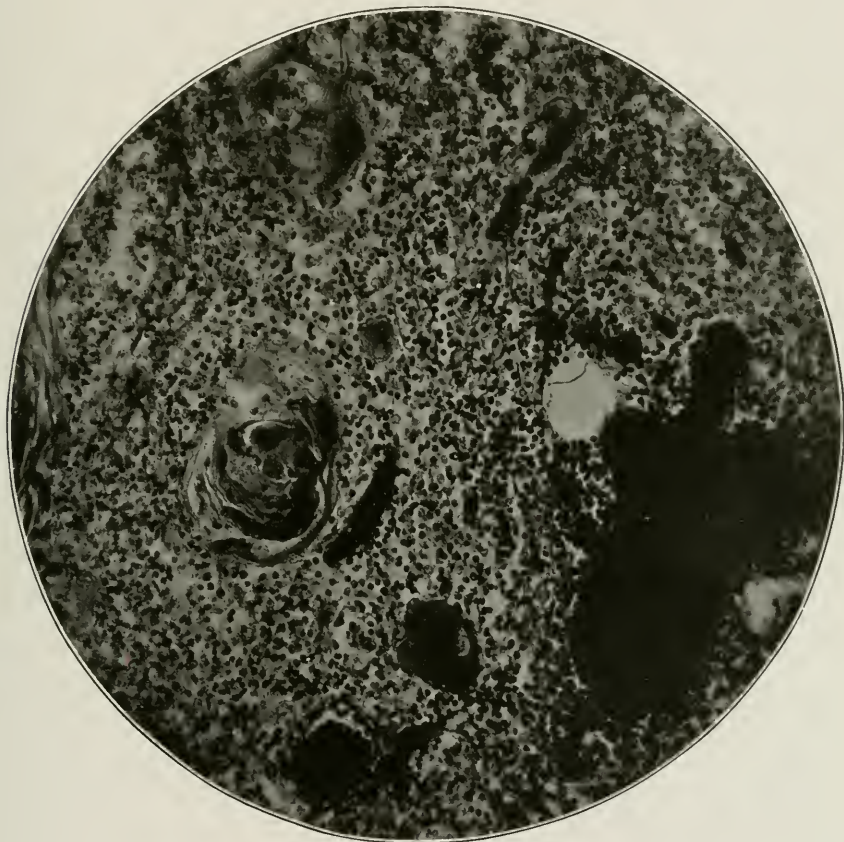


Fig. 8.—Section of the subinvolted thymus showing obliterating small blood vessels. They are in appearance not unlike Hassall's corpuscles.

patches, irregularly distributed. These patches bore no relation to the coronary or smaller vessels. The coronary vessels showed no pathologic change.

The thymus was large, covering almost the entire upper half of the pericardial sac. It was quite solid, about 1.5 cm. thick and rich in lymphoid tissue. The thyroid gland, although somewhat large, showed no gross pathologic change. The stomach and small intestines were normal. The large intestine was distended by solidified feces. The pancreas was normal, the liver markedly fatty, the kidneys congested, the testes and brain normal.

Microscopic Examination.—The liver showed marked fatty infiltration, diffusely and equally distributed. The pancreas, spleen, kidneys and testes were normal. The thyroid showed a simple struma.

Preparations from the wall of the left ventricle showed many connective tissue scars, varying in size (Fig. 1). They had not involved the perimysium externum, but were apparently produced mainly by the proliferation of connective tissue in the perimysium internum, leading to the isolation of individual muscle fibers (Fig. 2) and fragmentation of muscle fibers (Fig. 3). In younger, advancing scars small round fixed connective "tissue cells" were seen in large numbers (Fig. 4).

Fat infiltration was found in the connective tissue scars and about the blood vessels (Fig. 5), as it was in the skeletal muscles. As the degenerative process and the process of fibrosis were relatively mild in the heart, so the fat infiltration was rather meager. A larger amount of fat was under the epicardium, but this cannot be considered entirely pathologic.

Another feature, not unlike the pathologic change in the skeletal muscles, was the accumulation of many small round cells in the connective tissue trabeculae, in the fibrous scars, and, in a few instances, in the proximity of blood vessels (Fig. 6). This must not be interpreted as an inflammatory reaction but, as suggested by Marinesco and others, as a reactive phenomenon to the degenerative changes in the neighboring muscle fibers. Changes in the muscle fibers themselves were also observed. They partook mainly of the character of edema and swelling of some fibers, atrophy of others, with hyalinization and fragmentation (Fig. 7). Aschoff bodies were not found in the cardiac lesion described.

Findings in the thymus were extremely interesting. Particularly striking was the large number of vessels, some almost completely occluded, others partially so, by proliferation of the endothelial lining, while still others were open, but clogged with degenerating endothelial cells (Fig. 8).

COMMENT

Apparently this proliferation of the endothelium simulates the process of obliterating endarteritis, and when this process is complete, it gives rise to structures not unlike Hassall's corpuscles. The scarcity of these corpuscles, and the abundance of the transitional changes in the obliterating process of blood vessels, in the presence of subinvolution of the thymus, suggests a probable derivation of Hassall's corpuscles from obliterating vessels and that such a process might be responsible for the normal involution of the thymus.

The appended table was prepared to assist in determining the relation of the heart changes in progressive muscular dystrophy to a possible acute intercurrent disease, to some past illness, and to the pathologic process causing death. The table includes all cases which I have been able to find in the literature. All have shown fairly definite myocardial disease, though varying in degree. Although in three cases of the group a history of acute infection is given, the infection was insufficient to explain the diffuse nature and the morphologic character of the myo-

cardial involvement. In the column of intercurrent disease we find in one case pulmonary tuberculosis; in another, "a pulmonary condition," and in two others, pneumonia, while in the balance of the group pulmonary edema and grave gastro-intestinal disturbance were given as

RELATION OF HEART CHANGES TO DISEASE AS SHOWN IN THE LITERATURE

Investigator	Age of Patient, Years	Previous Illness	Nature of Terminal Illness	Anatomic Findings in Organs Other Than Heart	Changes in Heart Muscle
Goetz.....	10½	Scarlet fever	Not given	Congested lungs, spleen, kidneys	Enlargement of heart; increase of subendocardial fat
Ross.....	12	Pneumonia	Diarrhea, vomiting, prostration	No other findings *	Fibrous patches in wall of left ventricle; muscle pale, friable, fatty
Middleton.....	13½	Not given	A pulmonary condition	Pulmonary tuberculosis	Heart small, flabby, pale
Cardarelli.....	15	Not given	Diarrhea, prostration	Ulceration of large intestine	Enlargement; increase in subepicardial fat
Sachs and Brooks	12	Typhoid	Pneumonia	Bronchopneumonia, enlargement of spleen	Fibrous patches in walls of left ventricle; muscle pale, flabby, easily torn
Marinesco.....	Child Age ?	Not given	Pulmonary tuberculosis	Not given	Fibrous patches in the wall of the left ventricle
Goodhart and Globus	18	Not given	Pulmonary edema; hydrothorax; hydropericardium	Hypostatic pneumonia; hydrothorax; hydropericardium	Fibrous patches in the cardiac muscle; muscle pale and flabby; increase in fat; increase in the interstitial tissue
Goodhart and Globus	16	Not given	Pulmonary edema; hydrothorax; hydropericardium	Hypostatic pneumonia; hydrothorax; hydropericardium	Fibrous patches in the cardiac muscle; muscle pale and flabby; increase in fat; increase in the interstitial tissue
Bunting.....	15	Not given	Not given	Chronic suppurative bronchitis; bronchopneumonia; acute splenic tumors	Chronic fibrous myocarditis (translucent fibrous scars in heart muscle)
Potter.....	15	Measles, whooping cough	Bronchopneumonia	Fibrinous pleurisy; purulent bronchitis; subacute nephritis; passive congestion and fatty infiltration of the liver	Heart muscle pale, flabby; muscle fibers swollen, showing fatty infiltration and loss of striations

* Ross describes purulent fluid in pelvis of the kidney; the latter, however, being otherwise normal.

a cause of death. Here, again, there is little reason to suppose that the terminal acute pulmonary conditions or the gastro-intestinal disturbance accounted for the lesions of the heart, which were indeed of a chronic nature. Still, it is rather significant that pulmonary conditions should be found so frequently as a factor in the terminal issue of a large

number of cases. It is more reasonable to believe that the cardiac insufficiency was responsible for the development of conditions leading to death.

In studying the reports of the findings in organs other than the heart, we find few pathologic changes which could in any way suggest an etiologic factor responsible for the heart lesions other than primary myopathy. The character of the lesion offers further support to this assumption, for it appears that we are dealing with the same pathologic process in the heart as is found in the skeletal muscles. It is true that the process in the heart muscle is not so far advanced as in the striated voluntary type. This is probably due to a certain amount of resistance or partial immunity on the part of the heart muscle to the morbid process. This resistance or immunity prevents the heart muscle from going beyond the stage of fibrosis. However, the disease of the heart muscle must have advanced sufficiently in the early stage of the disease to permit its possible detection at that time by modern (electrocardiographic) methods. Such studies may hereafter yield very interesting results.

CONCLUSIONS

1. The heart muscle does not entirely escape the myopathic process characteristic of progressive muscular dystrophy.
2. It shows changes of a milder degree than those found in the skeletal muscles.
3. A few cases have been reported in which definite myocardial changes were found, but they have not always been looked upon as a part of the generalized process.
4. Probably myocardial changes will be more frequently found in progressive muscular dystrophy if the heart is systematically studied in every case.
5. The involvement of the heart muscle plays an important rôle in the terminal course of the disease and is perhaps responsible for some of the conditions which lead to rapid and fatal issue.

DISCUSSION

DR. WALTER TIMME: There are cases of muscular dystrophy in which there is marked myocarditis demonstrable in life, and many cases in which it is not demonstrable until after death. A clergyman of 82 years, who had had the disease since infancy and who had to be carried to the pulpit, had no sign of myocardial disease. The dystrophy was present in fourteen descendants in three generations; in none of them was there myocardial disturbance. A more rapid form of the disease, described by Gowers, causes death from the sixteenth to the nineteenth year and fatty degeneration of the heart assumes marked proportions. I think this disease is not exclusively a muscular one. It is not a

pure myopathy, but the myopathies are the most outstanding feature. Other atrophies, other dystrophies are seen, but not always. There may be only slight muscle changes. But necropsy will reveal disturbances in the anterior horn cells and in Clarke's column, in the anterior gray substance, in Lissauer's zone, which are not at all correlative to the slight muscular disturbance. In other cases at necropsy there are no spinal cord changes, but there have been marked muscular dystrophies. I roentgenograph all my patients for thymus; a large proportion have persistent thymus, and certain ones show a pineal shadow in early life. Marburg has published a paper on early pineal disturbance, accompanied by abnormal fat deposits about the body. I think we are beginning to see this disease as one in which the degenerative process is dependent on deficiency disturbances, probably in the control of some one or a group of the internal glandular units.

DR. FOSTER KENNEDY: Dr. Globus said that a certain number of the patients studied had died from a water logging of the chest. It would be interesting to know whether this had caused the death of his patient. I saw a child 13 years old, who died six years later with pseudodiaphragmatic paralysis. She suffered acute cardiac dilatation for three weeks before death, and had hydrothorax. The acute cardiac dilatation was not the primary lesion, but I thought it was a consequence of the heart strain.

DR. GLOBUS: I agree that progressive muscular dystrophy is a more or less generalized pathologic condition. In one case I found marked fatty infiltration in the liver, with no other pathologic changes in that organ. It was diffuse and bore no definite relation to the vascular or hepatic system of vessels and ducts. Bunting reported changes in the musculature of the large blood vessels. I noted hydrothorax and hydropericardium in three cases. The excess of pericardial and pleural fluids is best explained by the changes in the heart muscle.

DR. FOSTER KENNEDY: My experience has been that changes in the anterior horn and Clarke's column are not common. The neural changes occurring in paralysis are identical with those occurring after amputation, brought about by disuse. I think the changes in the spinal cord are secondary to those in the muscle. If we are to consider this as primarily an anterior horn lesion and a minor muscular disease, we become extraordinarily fogged in our classification of the pathology. Dr. Timme's experience has not been mine.

DR. WALTER TIMME: Three cases were reported by Kollarit and two by Schultz. These necropsy findings showed changes in the anterior horn cells in some cases, but they were not comparable to those in poliomyelitis. The cells were smaller than normal, but not degenerated. There was some disturbance of the fibers of the anterior cord substance. There were smaller cells in Clarke's column than normal. However, these findings are not universal. In cases of widespread muscular dystrophy there were often very few small cells. The conditions appeared to be absolutely independent of each other. It seemed to be a general atrophic condition involving the cord cells, as part of the general process and not as a secondary condition.

DR. GLOBUS: Modern neuropathologists have to account for two types of lesion: degeneration and inflammatory conditions. Poliomyelitis is a purely inflammatory lesion. It may have secondary degenerative processes. The cord changes in progressive muscular dystrophy are certainly not inflammatory. Are they to be considered as degenerative? Every degeneration in the central nervous system is accompanied by changes in the blood vessels and immediate

vicinity. The modern pathologist in such instances need not study the parenchyma; he studies mainly the relations of the glial elements. In degenerative lesions of the central nervous system one must find cells carrying away the products of destruction in the cord. The modification in size or shape of a nerve cell is not necessarily a pathologic phenomenon—it may be due to technical errors in preparation of material for study. But the glial reaction, accumulation of gutter and other phagocytic cells in the adventitial spaces is evidence of a degenerative process. Such changes are not encountered in the spinal cord in progressive muscular dystrophy.

SEX DEVELOPMENT AND BEHAVIOR IN MALE PATIENTS WITH DEMENTIA PRAECOX

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SEX DEVELOPMENT

The observations of Marro and others¹ have indicated the frequent occurrence of developmental defects of the reproductive apparatus and the physical characteristics of sex in idiots, cretins, epileptic persons, criminals, and persons with mental disease beginning at puberty. In male patients, varicocele, undescended testicle, hernia, hypospadias, unusually large and small testicles, deficiency of pubic hair and beard, and disturbance in skeletal growth, were reported to be more frequent than in controls. These observations were made before there was much evidence to indicate that certain endocrine glands are in some way essential to the normal development and functional activity of the reproductive apparatus.

More recently Emerson has reported the frequency of a type of imperfect physical development called "status lymphaticus" in alcoholic patients² and in patients with dementia praecox.³ He found this type in 22 per cent. of 1,000 alcoholic and drug cases in the alcoholic wards at Bellevue Hospital. Many of these inebriates were psychopathic. The incidence of status lymphaticus in the general medical wards was "less than a quarter of that found among alcoholics." He found the same type of physical make-up in 29 per cent. of 208 patients with dementia praecox from 16 to 50 years of age examined at the Manhattan State Hospital. Davis⁴ found the same type in 23.68 per cent. of 119 cases of war psychoneurosis, but in only 12.6 per cent. of 119 surgical cases in soldiers presenting no psychoneurotic features.

The essential features for the clinical recognition of "status lymphaticus" refer to the physical characteristics of sex. Yet 13 per cent. of the alcoholic persons classed by Emerson as normal had the feminine type of pubic hair, and in 24 per cent. the testicles were noted as small. This suggests the extent to which the opinion of the

1. Marro, A.: *La Puberté*, Paris, 1902. Hall, G. Stanley: *Adolescence*, New York, D. Appleton & Co., 1911, vol. 1, chap. 6.

2. Emerson, Haven: *Status Lymphaticus in Adults, Its Clinical Diagnosis and Importance*, Arch. Int. Med. **13**:169, 1914.

3. Emerson, Haven: *Note on the Incidence of Status Lymphaticus in Dementia Praecox*, Arch. Int. Med. **14**:881, 1914.

4. Davis, T. K.: *Status Lymphaticus: Its Occurrence and Significance in War Neuroses*, Arch. Neurol. & Psychiat. **2**:414 (Oct.) 1919.

examiner must enter into the classification of persons into physical types, especially when several features are considered which are not accurately defined or measured.

In the absence of well recognized and clear cut types of physical make-up, and in view of our still limited knowledge as to the exact relation between the various endocrine glands and the development of certain tissues and structures, it would seem better to study and classify these various physical features as separate units. Even in attempting to make a rough classification of individual parts, like the testicles, as to their development, the lack of a standard, the difficulty of exact measurement and the opinion of the examiner make accurate classification difficult. This is as true for controls as for the cases being studied.

TABLE 1.—SEX DEVELOPMENT IN DEMENTIA PRAECOX

Age on First Admission	Number of Cases	Size of Testes				Femine Pubic Hair, Percentage	Deficient Beard, Percentage	Scrotal Implantation, Percentage	Patients with Undescended Testis
		Patients with Both Testes Smaller than the Average, Percentage	Patients with One Testis Smaller than the Average, Percentage	Patients with Both Testes Larger than the Average, Percentage	Patients with One Testis Larger than the Average, Percentage				
16-20, under 21 when examined	47	6.3	6.3	14.8	6.3	12.7	61.7	27.6	3
16-20, 21 or over when examined	53	3.8	9.6	32.6	19.2	13.4	34.6	23.0	1
21-25	95	5.2	12.6	21.0	6.3	4.2	26.3	17.8	1
26-30	51	5.8	13.7	21.5	11.7	0.0	11.7	23.5	1
31-35	45	2.2	15.5	20.0	15.5	2.2	8.5	4.4	2
36-40	34	5.8	5.8	5.8	20.5	2.9	2.9	11.7	2

Case Material.—In Table 1 certain findings in 325 male patients with dementia praecox have been tabulated according to the age at the time of first admission to a hospital for mental disease. The first group of forty-seven patients, from 16 to 20 years on first admission, were examined before they were 21 years of age. Eleven were 20 at the time of examination and the others were under 20. The remaining 278 patients in Table 1 were considered as having chronic cases, and from one to twenty years had elapsed between their first admission and the time of examination. All were 21 or over at the time of examination. The age on first admission was unknown to the examiner at the time of examination, being determined later. The cases were unselected, and no previously recognized cases of endocrinopathy were included.

Size of Testes.—Testes have been classed as average, large or small, according to the examiner's conception of the average normal adult size. This conception was based on examinations of patients in the

wards of a general hospital and on a clay model having the dimensions given in Cunningham's anatomy. Those testicles which seemed by palpation to be definitely larger or smaller than the average were so classed, but no attempt was made to decide in each case whether the variation was sufficient to be called abnormal. The figures, therefore, merely indicate the percentage of cases showing variation from the average; they do not attempt to represent the percentage of cases showing abnormality. By this means, and with all examinations made by one observer, the error due to the examiner's opinion in each case is reduced to a minimum. This method affords a satisfactory working standard for comparing the size of the testes according to the age of the patient on first admission. The results are so arranged in Table 1.

The testes of patients first admitted between the ages of 16 and 20 compared favorably in gross size with those of patients first admitted between the ages of 21 and 40. Small testes were no more frequent in patients admitted at an early age than in those admitted later in life. Large testes were nearly as frequent in patients admitted and examined between 16 and 20 as in those admitted between 21 and 40, and were more frequent in those admitted between 16 and 20 but not examined until they were 21 or over. In explanation of the latter observation two possible factors may be considered, one or both of which may have been operative. First, in some cases with the psychosis developing during puberty there may have been an overgrowth of testicular tissue which was not completed until 21 or later. Second, there may have been an accumulation in the wards for chronic cases of patients with large testes. The prognostic significance of disturbances of sex development with regard to ability to leave the hospital and to rapidity of deterioration cannot be determined until consecutive early admissions have been followed for several years.

The majority of the patients examined had testicles which compared favorably in size with the normal, according to the examiner's conception of the normal average size. This was found to be true even for patients admitted and examined when they were between 16 and 20 years of age. In less than 5 per cent. of all patients both testes were noted as small. Typical infantilism was not encountered, but in a few cases both testes and penis were quite small and pubic hair almost totally absent. Typical infantilism has been observed in two patients first admitted before 16 years of age, but not included in the series. In several patients one testis was small while the other was large, and when one testis was undescended the other was usually as large or larger than the average.

In these examinations it was frequently observed, especially in young patients, that the testis felt unusually hard or firm or rubbery. In many such the epididymis and vas could be palpated only as a cord

firmly adherent along the border of the testis, so that it could not be separated out and palpated between the fingers. Many of these rather firm testes were not of the normal shape, but were nearly round or had a deficiency of tissue at the lower pole. A ridged or corrugated surface has been rather frequently observed. These findings suggest the possibility of a growth or overgrowth of tissue without proper differentiation, or an excess of connective tissue.

In view of the reports⁵ of failure of spermatogenesis and degenerative changes in the gonads in dementia praecox, it might be expected that testes smaller than the average would be rather frequent. Tiffany noted, however, that parenchymatous degenerative changes were accompanied by increased connective tissue even in young subjects, and that size and consistency cannot be taken as absolute criteria of the extent of degenerative changes. It might also be expected that patients with a long duration of the psychosis would show smaller testes than recent admissions with a short duration. In twenty-two patients between 16 and 40 years of age when first admitted, there were twenty-eight testes classed as small. Eleven were in patients who had been in the hospital more than ten years, and seventeen were in patients who had been in the hospital less than ten years. However, patients in whom degenerative changes would be most definite and rapid might not live long enough to be in the hospital ten years, or increase of connective tissue might compensate for parenchymatous shrinkage and so prevent decrease in gross size.

Feminine Type of Pubic Hair.—Three types of distribution of pubic hair have been observed: feminine, semifeminine and masculine. Only such cases were classified as feminine as showed the hair growth to be definitely limited to the pubic area with the distinct horizontal limitation and no hair extending along the midline toward the umbilicus. This type is shown in Figures 1 and 2. It is to be noted that the feminine type is an average growth limited to the feminine distribution rather than such a degree of deficiency as to limit it to this area. The comparatively few cases of definitely deficient pubic hair usually showed the masculine type on close examination. It was frequently observed, however, that with a good growth of the feminine type there was little or no hair on the scrotum and perineum.

5. Obregia, M.; Parhon, C., and Mechia, C.: Recherches sur les glands génitales testicules et ovaires dans la demence precocce, *L'Encéphale* **8**:109 (Feb.) 1913. Todde: Ricerche sulla funzione e sulla struttura della ghiandole sessuali Maschili nelle Malattie Mentali, *Riv. sper. di freniat.* **40**:233, 1914, cited in *Sex Organ Changes in Insanity*, editorial, *J. A. M. A.* **65**:254, 1915. Mott, Frederick W.: Normal and Morbid Conditions of the Testes from Birth to Old Age in One Hundred Asylum and Hospital Cases, *Brit. M. J.* **2**:655 (Nov. 22) 1919. Tiffany, W. J.: Pathological Changes of the Testes and Ovaries in Dementia Praecox, *New York State Hosp. Quart.* **6**:159 (Feb.) 1921.

The pure feminine type occurred in 13 per cent. of patients first admitted between 16 and 20 years of age, and in only 2.6 per cent. of those first admitted between 21 and 40. The difference is quite definite, since the type was accurately defined. That the feminine type is not merely a phase of male development is indicated by its occurrence in 13.4 per cent. of those first admitted between 16 and 20, but who were

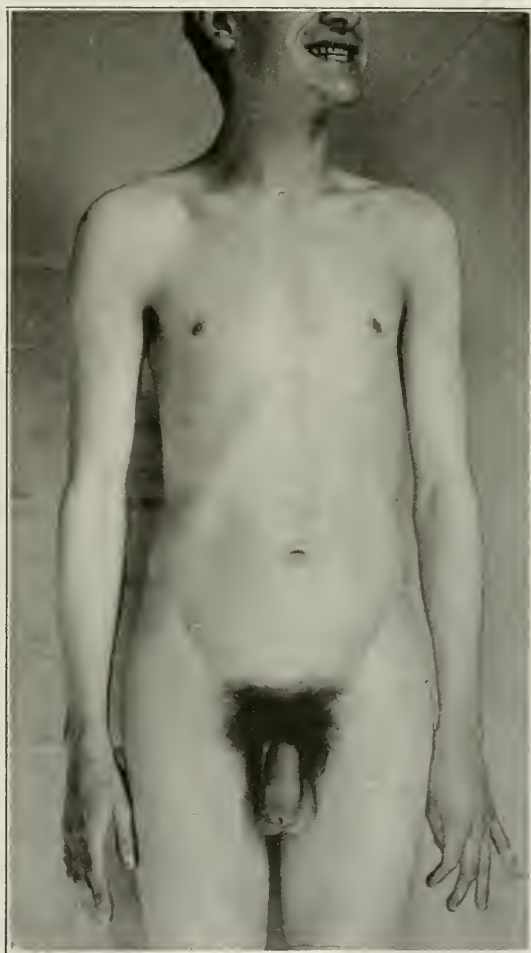


Fig. 1.—Patient with dementia praecox. Age on admission, 18; present age, 23; feminine pubic hair; deficient beard; no hair on the trunk; scrotal implant; testes average size; long thorax.

not examined until after 21. Their average age at the time of examination was 23 years.

The semifeminine type gave the impression of a primary feminine growth with the addition of a few hairs along the midline or arranged

in a whorl over the abdomen, the horizontal demarcation remaining distinct. This secondary growth was usually of much finer and shorter hair than that over the pubis. This semifeminine type was noted in about 11 per cent. of the patients examined, and was fairly evenly distributed through the age groups. Cases of this type are shown in Figures 3, 4 and 5.

Small testes were associated with the pure feminine type of pubic hair in only four of nineteen patients, and large testes occurred in four.



Fig. 2.—Patient with dementia praecox. Age on admission, 18; present age 23; feminine pubic hair; deficient beard; no hair on trunk; testes large; large bones and muscles.

In only two patients were both testes small. In two patients one testis was undescended, and in one of these the other testis was unusually large. Likewise in patients with definitely deficient pubic hair of the masculine type, there was no particular deficiency in the size of the testes.

Because of its persistence after puberty, the abundance of its growth and the size of the testes, it would seem that the greater frequency of

the feminine type of pubic hair in patients with early onset is something more than a retarded phase of normal male sex development. It seems more definitely a perversion or unevenness of development.

Deficient Beard.—Marro found absence of the beard in 13.9 per cent. of 395 criminals over 20 years of age, and in only one of sixty-three normal subjects. He also observed that absence of the beard was



Fig. 3.—Patient with dementia praecox. Age on admission, 19; present age, 24; eunuchoid body contour; semifeminine pubic hair; testes large.

frequent in the insane, and quotes Clouston as having observed that rapid growth of the beard was a favorable prognostic sign in the insanity of adolescence.

In the patients tabulated in Table 1 as having a deficient beard, there was a total absence or marked deficiency of hair on the sides of

the face and jaw, under the chin and on the neck. The hair that grew over this area was present in small patches or tufts separated by much larger areas without hair. A distinction is to be made between the hair on the lip and chin and that on the jaws and neck. The latter is more characteristic of the male. The former is frequent in females after

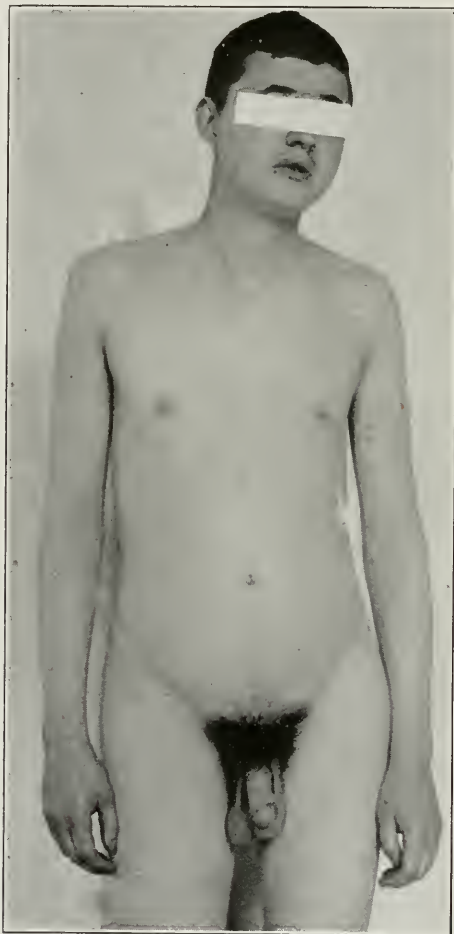


Fig. 4.—Patient with dementia praecox. Age on admission, 16; present age, 21; semifeminine pubic hair; absence of beard; scrotal implant; testes large.

removal of the ovaries and after the childbearing period. Martin⁶ has designated the lip and chin hair as primary and that on the jaws as secondary. In Figure 6 is shown a two weeks' growth of beard which

6. Martin, Rudolph: *Lehrbuch der Anthropologie*, Jena, 1914, p. 373.

is confined to the lip and chin with no hair on the jaws and neck. This absence is also shown in Figures 2 and 4, which were also two weeks' growths.

From Table 1 it will be noted that deficient beard was quite frequent in patients admitted at an early age and gradually decreased in frequency in patients admitted at a later age. A scant growth of beard would be expected in the group examined during puberty, but it will be noted that 34.6 per cent. of those admitted early still showed the deficiency when they were 21 or over. In the group of patients between 21 and 25 years of age on first admission there were forty-six who



Fig. 5.—Patient with dementia praecox. Age on admission, 19; present age, 30; semifeminine pubic hair; scrotal implant; testes quite large; actively homosexual.

had been in the hospital long enough to reach the "chronic" wards. Ten of these forty-six, or 21 per cent., showed deficient beard after they had reached an average age of 25.8 years. Five of the ten were over 26.

Small testes were associated with deficient beard in only a few cases, being noted in only two of the eighteen patients with deficient beard in the 16 to 20 group examined when they were 21 or over. In ten of these eighteen patients, one or both testes were classed as large. Small testes were noted in only two of the ten patients with deficient beard in the 21 to 25 group who were examined after they had reached an average age of 25.8 years.

Scrotal Implant.—Patients were classed as having a scrotal implant when a fold of the scrotum on each side extended above and around the penis so that the penis seemed to emerge between these folds (Figs. 1, 4 and 5). Scrotal implant occurred rather frequently, as shown in Table 1, and was somewhat more frequent in patients admitted at an early age than in those admitted at a later age. This relation to age was not as definite, however, as that of the secondary sexual hair. This anomaly of genital development probably should not be classed as a secondary sex character in the same category with the secondary sexual hair and having the same relation to the gonads.



Fig. 6.—Patient with dementia praecox. Same as Figure 1. Two weeks' growth of beard; absence of hair from neck and sides of face.

SEX BEHAVIOR

An attempt has been made to approach the problem of sex in dementia praecox from the standpoint of behavior, somewhat according to the conception of behavior outlined by Watson.⁷ I have attempted, first, to determine what the actual sex behavior of persons developing dementia praecox has been. It is well recognized that in many cases the sex life has been abnormal, and that a sexual coloring is often

7. Watson, John B.: *Psychology from the Standpoint of a Behaviorist*, Philadelphia, J. B. Lippincott Co., 1919.

prominent in the clinical picture of the psychosis. However, no systematic observations seem to have been made to determine just what these persons have actually done in the way of sex behavior, how far they have advanced in the evolution of their sex life, whether or not an adult and socialized level of sex behavior has been reached, and whether this behavior at the adult level has been maintained.

Marriage in Dementia Praecox.—While marriage cannot be taken as an absolute criterion of the complete evolution of behavior in the realm of sex, yet the low marriage rate in males developing dementia praecox is rather striking. Pollock and Furbush⁸ report 2,232 male first admissions with dementia praecox in twelve states in 1919. Of these, 73.9 per cent. were single. From Table 2 it will be seen that the marriage rate for males developing dementia praecox is definitely below that of the general population, and that the difference is greatest in

TABLE 2.—MARRIAGE IN RELATION TO AGE

Dementia Praecox				General Population *	
Age on First Admission	No. of Cases	Number Married	Married,† Percentage	Age	Married,† Percentage
15-19.....	76	1	1.3	15-19	0.6
20-24.....	108	1	0.9	20-24	20.9
25-29.....	52	6	11.5	25-34	63.3
30-34.....	57	16	28.0		
35-39.....	34	14	41.1	35-44	82.7
40-44.....	16	11	68.7		
Total.....	343	49	14.2		

* Middle Atlantic Division of United States, 1910.

† Widowed and divorced included.

the younger age groups. The dementia praecox patients referred to in Table 2 are those in whom physical sex development was studied.

Relations with the Opposite Sex.—More definite and concrete evidence of the degree of maturity attained in sexual life would be that of adult relations with the opposite sex. This question has been investigated in 137 unselected cases. The results are shown in Table 3. When the patient was mute, refused to answer, or if the answers were indefinite, evasive, or not considered dependable, the case was placed in the "not determined" group. In those indicated as "inadequate" one or more attempts at adult relations had been made with more or less success, but this level of behavior had not been established and maintained, for which various reasons were given. In those indicated as "adequate for a time," adult relations had been established and kept up for varying periods. In many of these, adult relations had not been indulged in for some months or years prior to first admission. The

8. Pollock and Furbush: Mental Diseases in Twelve States, 1919, Ment. Hyg. 5:353, 1921.

relation of this failure to the onset of psychotic symptoms is being made the subject of further observations.

Table 3 shows that 120 patients who were first admitted between 15 and 34 years of age answered the inquiry in a satisfactory manner. Of these 120 patients, 64.1 per cent. had never had sexual relations with the opposite sex. In an additional 15 per cent. relations had been attempted and carried out one or more times but had not been satisfactory, or for some other reason this form of sexual activity had been abandoned. Fear seemed to condition the reaction abnormally in certain of these cases, with a resulting panic.

Only 20.5 per cent. had ever reached an adult level of behavior and maintained it for even a short period either single or married, assuming that relations were kept up in all those who married. More detailed inquiry in several cases of the 12.5 per cent. of single persons who had kept up relations showed that such relations had usually been on the lowest prostitute basis. No further interest in the partner was

TABLE 3.—SEX BEHAVIOR IN DEMENTIA PRAECOX

Age on First Admission	No. of Cases	Activity Not Determined, Cases	Single				Married, Cases
			No Relations with the Opposite Sex		Relations Inadequate, Cases	Relations Adequate for a Time, Cases	
			Cases	Percentage			
15-19.....	30	3	22	81.4	4	1	0
20-24.....	53	10	29	67.4	6	7	1
25-29.....	34	3	17	54.8	5	4	5
30-34.....	20	1	9	47.3	3	3	4
Total.....	137	17	77	64.1	15%	12.5%	8.3%

manifest, and masturbation was practiced in the intervals. Even common law relations were avoided or not kept up. Heterosexual life in any socialized way seemed just out of reach. In this sense the behavior could scarcely be called adequate.

Early onset of the psychosis is the first point for consideration in explanation of these findings. The cases have been tabulated according to the age at the time of first admission rather than the age of onset of the psychosis. By careful study of these cases it might be found that the actual onset had been sufficiently early to explain the lack of sexual activity. It will be observed from the tables that the earlier the age on admission the higher was the percentage of cases showing sexual inactivity. But even in patients first admitted between 25 and 34 years of age there had been no relations with the opposite sex in over 50 per cent. To explain these simply on the basis of early onset, it would be necessary for the onset to have occurred from five to ten years before the first admission of the patient. This would indicate that the onset occurred at puberty or soon after, for it can probably be safely assumed

that most males accomplish heterosexual relations soon after puberty and usually repeat such relations at more or less frequent intervals. It would, therefore, seem that the findings cannot be easily or simply explained on the basis of early onset of the psychosis.

It did not appear that genuine moral or religious self-restraint had an influence, except possibly in a few cases, and in only a few were any such reasons offered. The frequency of masturbation and anti-social and immoral behavior often displayed by these patients speaks against such an explanation. A rather marked interest in religion is frequently manifested by persons developing dementia praecox. In many such cases it seems rather apparent that such abnormal interest is a part of an inadequate personality, or a kind of compensation for a sense of inferiority.

Further studies of sex from the standpoint of behavior are being made with an attempt to estimate, even in a rough way, the sex equipment of the individual in the way of instincts and emotions and of physical sex growth, to follow the evolution of the sex behavior reactions with this endowment, and to ascertain what conditioning factors may have played a part. A perfectly constructed biologic mechanism, with complete instinctive and emotional equipment, and capable of functioning at the normal adult level of efficiency, has not been assumed, for it seems quite possible that such biologic factors as growth, metabolism and the internal secretions may play a part in determining sex behavior, and other behavior as well.

DISCUSSION

The findings indicate a disturbance of sexual development and a failure of sexual maturity which is most marked in patients admitted to the hospital during the years of puberty and adolescence. Patients admitted at an early age give the impression of an unevenness of sexual growth, the development of the secondary sexual characters lagging behind that of the testicles. The association of well developed genitalia with deficient secondary sexual hair in patients well past the age of puberty is shown in the photographs. These patients seem to lack the finishing touches of complete physical maturity.

The findings cannot be easily interpreted in terms of internal secretions. Gonads, and especially interstitial cells, are essential to complete sex maturity, but they are not self-sustaining. Other internal secretions may stimulate or activate them. Of interest in this connection is the patient shown in Figure 3, who had the physical make-up of the eunuch associated with well developed testicles.

Further evidence of a failure of complete sexual maturity, especially in terms of functional integration of the organism, is the large number of dementia praecox patients who have failed to reach the adult level

of sex behavior. That this failure is due to biologic inadequacy limited to the sex mechanism probably should not be inferred. The total functional inadequacy of many of these patients is seen in the well recognized inadequacy of their behavior in other directions, and the sex mechanism does not generate its own power, but derives it from the common source and merely transforms it to its own use. Adequate functional activity of the pituitary, thyroid, and suprarenals seems to be necessary for sex growth and function.

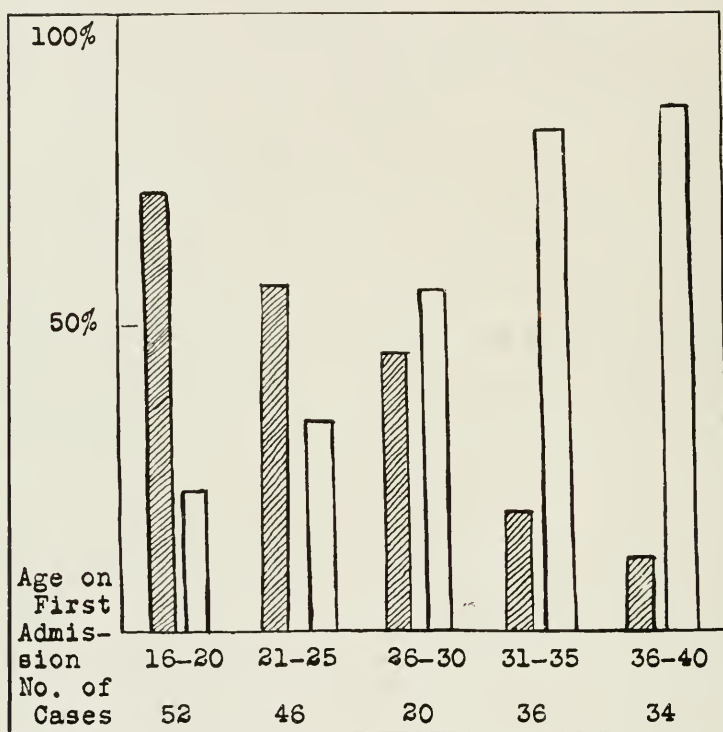


Fig. 7.—Diagnosis in relation to age. The dark areas represent catatonic and hebephrenic types, the light areas paranoid types.

The greater frequency of imperfect sexual maturity in young patients correlates well with the clinical types which are more frequent in early admissions. This relation in one series of patients included in this study is shown in Figure 7.

SUMMARY OF FINDINGS

1. The testicles of dementia praecox patients first admitted to the hospital between 16 and 20 years of age compared favorably in size with those of patients first admitted between the ages of 21 and 40.

2. Pubic hair of a definitely feminine distribution was present in 13 per cent. of patients first admitted between the ages of 16 and 20, and was still present in 13.4 per cent. after they were 21 years of age or over, but was present in only 2.6 per cent. of those first admitted between the ages of 21 and 40.

3. A definite deficiency of beard occurred more frequently in patients admitted early than in those admitted late. This deficiency persisted until after 21 in 34.6 per cent. of those first admitted between the ages of 16 and 20, and was still present in 21 per cent. of patients first admitted between the ages of 21 and 25 when those showing the deficiency had reached an average age of 25.8 years.

4. Deficient development of the secondary sexual hair did not depend on the size of the testicles, being associated with rather large testicles as frequently as with small ones.

5. The marriage rate of males developing dementia praecox was definitely below that of males in the general population.

6. Adult sexual relations with the opposite sex had never been accomplished by 64.1 per cent. of 120 dementia praecox patients who answered the questions in a satisfactory way.

7. Only 20.5 per cent. of the patients had reached an adult level of sex behavior and maintained it for even a short time, either married or single.

SPIROCHETE STAIN IN MULTIPLE SCLEROSIS

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During the last ten years there has been an increasing interest in the etiology of multiple sclerosis. This has been stimulated by a growing body of facts which leads away from endogenous or idiopathic conceptions of this disease and gives increasing evidence of a living causative agent. Much has been written in reporting attempts to cultivate or demonstrate some organism by inoculation, and interest has centered about a spirochete as a possible cause of this disease. Animal inoculations have been made, and human tissue has been examined. A number of positive results have been obtained, but, in part these so disagree among themselves as to the characteristics of the suspected organism that different organisms must have been dealt with. Hauptmann¹ has given such a comprehensive review and criticism of the work reported previous to 1921¹ that further details will not be given here. Several reports have appeared since then. Birley and Dudgeon² have been unsuccessful in all attempts to isolate an organism or to transmit the disease to animals. They, however, feel that the evidence favors the inflammatory conception of the disease. Magnus³ has also been unsuccessful in animal inoculation experiments. Guy,⁴ who was the pioneer in experimental investigation of the disease, has done further work which makes him feel safe in concluding that multiple sclerosis is an infectious disease whose virus may sometimes be found in the cerebrospinal fluid. Schuster⁵ has recently announced the first demonstration of spirochetes in sections of human tissue. He used Jahnel's stain modified. His evidence in support of this claim is not convincing.

Since Jahnel⁶ through his modifications of previously existing methods of spirochete staining has so established the procedure as to make it dependable, it was considered worth while to undertake the study of a number of sections from cases of multiple sclerosis by his method.

1. Hauptmann: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **70**:300, 1921.

2. Birley, J. L., and Dudgeon, L. S.: *Brain* **64**:150, 1921.

3. Magnus, V.: *Etiology of Disseminated Sclerosis*, abst., *J. A. M. A.* **77**: 1012 (Dec. 17) 1921.

4. Guy, W. E.: *Brain* **64**:213, 1921.

5. Schuster, J.: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **73**:433, 1921.

6. Stevenson, G. S.: *Two Recent Improvements in the Staining of Spirochetes in Nervous Tissue*, *Arch. Neurol. & Psychiat.* **7**:349 (March) 1922.

Four cases were available for this study. All four had been shown by microscopic examination to possess the typical lesions of multiple sclerosis. In one case recently acquired by us only a few sections had been made. In all thirty-seven sections were examined; in Case 1 ten sections, Case 2 twelve sections, Case 3 eleven sections, Case 4 four sections. Sections were taken from many parts of the cortex, brain stem and spinal cord and one from the internal capsule. A few were taken with reference to an active process found by other stains. Some such process was present in twelve specimens examined. Most sections were taken at random and from widely scattered parts. These tissues were stained in four lots at different times. Three of these lots also included control tissue from parietic brains known to contain spirochetes. In each instance a good spirochete stain of this control tissue was obtained. In studying these tissues one section of each block was examined thoroughly and completely by the aid of a mechanical stage. High-dry magnification was mostly used. Whenever any suspicious object was seen it was examined more thoroughly with the oil immersion lens.

In all these sections no object was found which could be taken for a spirochete.

SUMMARY AND CONCLUSIONS

In the study of thirty-seven sections from four cases of multiple sclerosis stained by Jahnke's new method no spirochetes were found.

These studies indicate that spirochetes are absent or infrequent in multiple sclerosis tissue.

Spirochetes held to be associated with multiple sclerosis, if present, probably cannot be demonstrated by the most dependable silver stains now available.

Abstracts from Current Literature

THE DIAPHRAGM CENTER IN THE CEREBRAL CORTEX, AND HICCUP. KNAPP, Monatschr. f. Psychiat. u. Neurol. **50**:333 (Dec.) 1921.

The ordinary movements of the diaphragm as they occur in respiration and phonation, beginning at birth are under the control of subcortical motor centers. Gradually, the cerebrum acquires a degree of control over these lower centers, as evidenced by the fact that the normal reflex automatic movement of the diaphragm can be to some extent voluntarily controlled; we can voluntarily increase or decrease our respiration, or for a brief period, even stop it altogether. Thus on physiologic grounds we are justified in assuming a cortical center for diaphragmatic movements. This center is intimately related functionally to the cortical centers for respiration and phonation; but they cannot be identical, as each can act independently of the others.

The diaphragm is a bilaterally acting muscle. It has been demonstrated for other muscle groups of this type (phonation, etc.) that there is bilateral cortical representation and bilateral control. Unilateral stimulation produces a bilateral effect, while unilateral destruction produces no effect, or a transient and incomplete paresis. So we may assume that the diaphragmatic center is a bilaterally represented and bilaterally acting center, each hemisphere having stronger influence over the contralateral muscle. One would therefore expect *a priori* an irritation of the diaphragmatic center in one hemisphere to produce a bilaterally symmetrical diaphragmatic contraction. Clinicopathologic observation shows that this is the rule. Exceptions to this rule occur (rarely) in both directions, irritation of one center producing contraction only of the contralateral half of the diaphragm, and destruction of one center producing contralateral paralysis.

Bulbar Hiccup: In the majority of cases, hiccup is independent of the cerebral cortex, and is dependent on direct or reflex stimulation of the centers in the medulla which control the automatic and reflex diaphragmatic movements. As an example of this type, we can consider hiccup resulting from an overloaded stomach. The dilated stomach presses on the sensory plexuses above and below the diaphragm, and sends sensory impulses up to the medullary centers and out by way of the phrenic. Sensory stimuli of this kind can come either by way of the vagus, or the sympathetic, nerve. In this general group belong the cases of hiccup due to disease of the stomach and bowel, to ileus and peritonitis, to disease of the uterus and adnexa, following kidney and prostate operation. Hiccup occurring in crises in tabes dorsalis may be due to direct irritation of the ninth and tenth nuclei in the medulla. Hiccup due to bulbar irritation may also occur in the course of cerebral diseases, such as tuberculous meningitis. Bruns mentions it in tumors of the medulla and tumors of the posterior cranial fossa. In all these groups of cases we have direct irritation of the medullary center.

Cortical Hiccup: In another group of cases, hiccup is a cortical focal symptom, due to the irritation of the diaphragmatic center in the cortex. The author reports four cases of his own and four from the literature. This type of hiccup is analogous to jacksonian epilepsy, and in four of the cases the diaphragmatic contraction alternated with contractions of other muscle groups of jacksonian type.

Prognosis: Formerly hiccup occurring in cerebral disease was considered a serious symptom. This is true of cerebral hiccup of bulbar origin, but is not so true of the cortical type.

Localization of Cortical Hiccup: Bergmark has reported a unilateral crossed paralysis of the diaphragm where the focus lay in and in front of the precentral gyrus. In his case the lesion extended from the top of the hemisphere to the upper part of the arm center. Therefore the diaphragm center must be between the leg and arm regions, where Sherrington puts the trunk zone, or in front of the leg and arm region, where most other investigators put the trunk centers. In some previous studies the author demonstrated that the arm and leg center touch, there being no room between them for the trunk centers. Therefore he believes the trunk and the diaphragm centers lie in front of the arm and leg centers where the centers for hip and shoulder meet. The weight of evidence from the whole group of cases is that the cortical centers for the diaphragm lie in the upper half of the foot of the second frontal convolution. According to an observation of Bechterew, the respiratory center lies very close to this same region.

Not all the cases corresponded to this localization. In one case the lesion involved the foot of the central convolution, and in two others the temporal lobe, especially the anterior portion. For this group the author does not assume a second center. He believes that in all this group, directly or by pressure, there is involvement of the foot of the precentral gyrus. Here lies the center for phonation, which is intimately associated with diaphragmatic movements, and which must be connected with the diaphragmatic center by extensive association tracts.

Thus we have two cortical zones from which a contraction of the diaphragm can be produced. The first lies in the foot of the second frontal convolution, in front of the lower part of the leg center and the center for shoulder movements, close to the center for the abdominal muscles and in the region of the inspiratory (respiratory) center.

A second center lies a considerable distance away. It is closely connected with the centers for phonation, chewing and swallowing, in the foot of the precentral gyrus, close also to the center for facial movements—all centers which have close relationship to the diaphragmatic and respiratory movements.

The first is undoubtedly the true cortical diaphragmatic center. The second is connected with this with numerous association fibers.

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STUDIES IN EXPERIMENTAL TRAUMATIC SHOCK. V. THE CRITICAL LEVEL IN A FALLING BLOOD PRESSURE. W. B. CANNON and McKEEN CATTELL, Arch. Surg. 4:300 (March) 1922.

The authors begin this study by noting that in forty-five coincident determinations of blood pressure and carbon dioxid capacity, made on wounded men, the alkali reserve decreased with the falling arterial pressure. The respirations increased in rate as the alkali reserve diminished; they were, however, shallow and feeble, and in the authors' opinion were probably not effective in ventilating the lungs and thus lessening the concentration of carbon dioxid in the blood. The primary factor in the production of the low carbon dioxid capacity, the authors believe, is the marked fall of arterial pressure with a resulting diminution of the oxygen supply.

Experimental work on animals under conditions analogous to those of shock is discussed. A small slit was made in the pericardium of the animal and a cannula connected with a funnel by a rubber tube inserted; by means of this arrangement, physiologic salt solution was allowed to flow into the pericardial cavity. The output of the heart was directly controlled by the pressure of the fluid in the pericardium, and thus by raising or lowering the funnel any desired arterial pressure could be maintained. Artificial respiration and ether anesthesia were used. The alkali reserve of the blood plasma was determined each hour at various pressures.

The results obtained by this method showed that with a pressure reduced to 80 mm. of mercury, the alkali reserve was unaffected after one hour; at 70 mm. there was a slight reduction, averaging 3 per cent.; at 60 mm., there was a marked fall; at 50 mm. there was a still greater fall. The most rapid fall occurred during the first hour, after which it reached a stationary low level. The alkali reduction rapidly disappeared when the blood pressure was allowed to return to normal. Four animals bled to 20 per cent. of their calculated blood volume to produce a condition analogous to hemorrhage, with blood pressures reduced to 80 per cent., showed indications of inadequate circulation, which was less when the blood pressure was raised to 90 per cent. The authors conclude that the critical level, that is, the point beyond which oxidation is inadequate, is 80 mm.; accordingly, reduction of the alkali reserve is not likely to appear. These observations are corroborated by clinical evidence.

Morphin was found to modify the development of acidosis. When morphin was given before the pressure was lowered to 60 mm., the alkali reserve was not reduced; when it was given after the pressure was lowered to 60 mm. the alkali reserve, which had fallen, began to rise, and after two hours was largely restored. The authors suggest that morphin reduces the activity of tissues, lessens the demand for oxygen, and thus compensates for the smaller supply of oxygen in the sluggish blood flow.

When the blood pressure was reduced 60 mm., held for an hour, by intra-pericardial compression, and then released, it rose to normal; when the procedure was repeated after an interval of an hour, the blood pressure did not rise so high, and after another hour it did not rise at all. This is interpreted to mean that injury to the vasomotor center has resulted, and after a prolonged low pressure, the maintenance of vascular tone has been impaired.

The authors review the previous work on the sensitiveness of various tissues due to a lack of oxygen. Muscle withstands anemia well; the brain is particularly sensitive. The pyramidal cells of the cortex are the most sensitive, chromatolysis being produced in eight minutes. The Purkinje cells of the cerebellum are next in sensitivity, while the medulla and sympathetic ganglions are more resistant. The work of E. L. Porter is cited, showing that the reflex arc disappears when the oxygen content of arterial blood is reduced to 4.5 per cent. by volume, and his conclusion that a lack of oxygen is more important than an accumulation of carbon dioxide in abolishing the capacity to transmit nerve impulses is noted. The authors quote these data with reference to their deduction that in the conditions of low blood pressure studied there is injury to the vasomotor center as a consequence of insufficient oxidation from a sluggish flow of blood.

The practical points following from the study are stated. The low blood pressure incident to shock must be combated at once. If the pressure falls below 80 mm., damage will result to the neurons, particularly in the vaso-

motor center; if the pressure is persistently low, the vasomotor center will be permanently damaged, and the pressure will remain low because of the lost capacity to maintain vascular tone.

VONDERAHE, Philadelphia.

CLINICAL STUDIES ON THE DEVELOPMENT OF SCHIZOPHRENIA.

I. ERWIN POPPER, *Monatschr. f. Psychiat. u. Neurol.* **50**:159 (Sept.) 1921.

The cause of schizophrenia is unknown, but certain factors contribute to its development which we can study. Considering the incidence of the disease, we find that beginning with the second decade there is a rapid increase in the number of cases, reaching a maximum at about the twenty-fifth year; after that it begins to fall, first slowly, then more rapidly. From the fortieth year on, the number of cases is small. If we separate the curves according to sex, we find that in the male the peak is a little higher, and the drop which follows the peak is sharper. After 40, it practically reaches the base line. In women the peak is not quite so high, and runs along fairly level from 25 to 35, dropping toward the base line more slowly. Practically all the cases occurring after the age of 40 are in women.

Hereditary Factors: A careful family history showed for 60 per cent. of the men and 61 per cent. of the women, cases of frank psychoses, marked anomalies of character, affectivity and general make-up "queer," psychopathic, neurotic, hysterical and alcoholic persons.

In studying the intellectual status of his schizophrenic patients, the author depends chiefly on the school record. The figures are as follows: Men: Intelligence good 36 per cent., medium 15 per cent., and poor 49 per cent. Women: Intelligence good or fair 66 per cent., poor 33 per cent.

Prepsychotic peculiarities of character, or affectivity, were shown by 43 per cent. of the men and 43 per cent. of the women. These figures are so high that they strengthen the view that preexisting affective anomalies are important factors in the development of schizophrenias.

Developmental anomalies on the somatic side occurred in 29 per cent. of the men and in 25 per cent. of the women.

Epilepsy and epileptiform symptoms of various kinds occurred in 15 per cent. of the men and 13 per cent. of the women.

Altogether, more than 60 per cent. of the patients showed hereditary factors, and 66 per cent. showed abnormalities of the prepsychotic make-up. Hereditary factors alone, without any abnormalities of prepsychotic development, occurred in 18 per cent. of the men and in 15 per cent. of the women. Prepsychotic abnormalities without any determinable hereditary factors occurred in 19 per cent. of men and in 17 per cent. of women.

In 15 per cent. of the men and in 22 per cent. of the women no hereditary factors and no prepsychotic abnormalities of character or disposition were found to determine the occurrence of the disease.

In considering the causation of schizophrenia, we have three possibilities: (1) that it is a purely exogenic disease; (2) that it is a purely endogenic disease; (3) that endogenic factors are essential, but that on these must be grafted certain exogenic causes.

Hereditary taint and abnormalities in the prepsychotic make-up are so frequent that we must recognize their importance in determining the development of schizophrenia. We realize that data of this type are more or less vague and ill defined. When we try to group them into a composite picture, we find that we cannot define a schizoid personality or a schizoid constitution. We

cannot select any one characteristic which would justify us in concluding that a person would become a schizophrenic. We cannot as yet recognize the disease in the making. And even when we accept these endogenic factors at their full value, we find that we must still attempt to explain the group of cases (one sixth of the total number) in which no such factors can be determined.

THE TRANSPLANTATION OF SKIN IN FROG TADPOLES, WITH SPECIAL REFERENCE TO THE ADJUSTMENT OF GRAFTS OVER THE EYES, AND TO THE LOCAL SPECIFICITY OF INTEGUMENT. WILLIAM H. COLE, J. Exper. Zool. **35**:353 (May 20) 1922.

Over the eyes of tadpoles of *Rana catesbeiana* and *R. clamitans* varying in length from 20 to 100 mm., opaque skin was transplanted to demonstrate the existence of any regulatory interaction between graft and skin tending to restore the function of the eye. In 66 per cent. of the operations in which tail skin was placed over the eye, the grafts were absorbed in a way which tended to expose the eyes. Back skin grafts were never so absorbed. Tail and back skin placed on other parts of the body were never absorbed. Tail skin grafts over hemispheres of glass or celloidin were absorbed in the same way. Back skin grafts so placed were not absorbed. Tail and back skin grafts over thin glass plates were not regularly absorbed. The foreign material is not the stimulus for absorption. The absorption over eyes and over the convex "artificial eyes" is caused by the curvature of the eyeball or the "artificial eyes." The more compact structure of back skin prevents its absorption. The curvature of the graft during healing causes a tension which is the mechanical stimulus for absorption. All tail skin grafts proliferated new tissue. The anterior and posterior ends of such grafts can regenerate a tail tip containing notochord and nerve cord. Following proliferation all grafts enter a state of equilibrium in which there is no increase in their size. There is no functional regulation of skin grafts over the eyes. This is the result of the high state of differentiation and specificity attained by the skin and the eye.

Integument from the tail, back or belly transplanted to another region of the same animal preserves its individual characteristics indefinitely. Homeo-transplants of skin preserve their individuality only temporarily, their tissues being replaced by regenerated tissue of the host. The integument of frog tadpoles is locally specific, and is self-differentiating when transplanted to new soil on the same animal.

Autotransplants of white belly skin on the back or tail acquire melanophores through the formation of pigment cells from epithelial cells in situ. Homeotransplants acquire melanophores through epidermal migration from surrounding skin, followed by formation of pigment in situ.

When the conjunctiva of frog tadpoles is extensively injured the regenerated tissue is pigmented, first by migrating epidermal melanophores and later by a mitotic multiplication of integumentary cells forming dermal melanophores and xantholeukophores. This is further proof that no correlation between the eye and the overlying integument exists in tadpoles 20 mm. or more in length.

Expansion of the dermal melanophores is caused by a low temperature, by darkness, by a 0.1 per cent. chloretone solution, by anoxemia and by the low metabolic rate coincident with a moribund state. Contraction is caused by a high temperature, by light, and by a return of normal environmental condi-

tions after chloretone or anoxemia. Contraction is also coincident with recovery from a moribund state. Expansion or contraction usually requires about thirty-six hours for completion. It is probable that any stimulus lowering the metabolic rate of the whole animal causes expansion, and that stimuli causing an increase in metabolic rate cause contraction of the melanophores.

WYMAN, Boston.

ELEMENTARY CONVULSIONS IN CATATONIA. BAUSCH, *Monatschr. f. Psychiat. u. Neurol.* 50:319 (Dec.) 1921.

Through the work of H. Fischer, study of the convulsion has become a problem of its own, independent of the disease in which it occurs. The convulsion is not pathognomonic of any specific disease, but represents a type of reaction to various exogenic and endogenic traumas. This reaction type—the convulsive faculty—is not dependent on the brain alone but on a mechanism preformed in the entire body, corresponding to the mechanism which determines the general motor reaction forms of the organism.

The position which Vorkastner takes illustrates the general attitude toward the cases of convulsive seizures occurring in catatonia. Vorkastner asks the following questions:

1. Is there a dementia praecox with symptomatic convulsions?
2. Is there an epileptic psychosis simulating catatonia?
3. Is there a combination of two psychoses—catatonia and epilepsy?

The author considers that this way of looking at the problem is faulty and of itself interferes with the solution. It is due, in considerable measure, to the use of the term "epileptic seizure" or "epileptiform seizure" for the convulsion, thus substituting the idea of a disease entity for a mere symptom. If we accept Fischer's thought, separating the convulsive seizure from the convulsive disease, and using a name—elementary convulsion—independent of any disease concept, we come to a different formulation of the problem.

1. Are the elementary convulsions a specific symptom of dementia praecox, more particularly the catatonic type; or (2) are we dealing with purely symptomatic convulsions, such as occur in other endogenic diseases (diabetes, exophthalmic goiter, etc.)?

Looking at it in this way, Vorkastner's questions 2 and 3 are automatically eliminated, and we can dispense with the idea of any such combination disease as catatonia-epilepsy (Vorkastner) or "epileptic form of catatonia" (Urstein). The author illustrates his point of view with two case reports of convulsive seizures occurring in cases of dementia praecox.

In the first case there was a family history of epilepsy (paternal grandmother), as well as certain stigmas during childhood, such as bed wetting, abnormal fears, etc. The patient was solitary, unadaptable and shy. At 25 the psychosis began, and developed into a typical catatonia. During the course of the disease, elementary convulsions occurred. The author believes that they are the result of a constitutional component.

In Case 2 there is no history of convulsive diseases in the family. The patient had convulsions in the first months of life, but otherwise developed normally. Change in character which represented the onset of his psychosis began at puberty. It developed into a typical dementia praecox (catatonia), during the course of which a number of elementary convulsions occurred. For a time these dominated the picture, being classed sometimes as hysterical, sometimes as epileptic. The whole personality gave no suggestion of an

epileptic character. On the other hand, the emotional dulling was progressive during the period of observation. Bausch calls attention to a curious variability in the motor manifestations of the disease, stupor and akinesia being replaced at various times by excitement and elementary convulsions. The convulsions occurred only at the beginning or end of a stupor period. Often they started a period of general motor excitement, with impulsive acts, verbigeration and lively hallucinosis. This relationship between cataleptic phenomena and stuporous conditions on the one hand, and convulsions and motor excitement on the other, points to a relationship between these various manifestations of motility.

In both cases, then, there is a constitutional convulsive factor (heredity in one, convulsion in early life in the other). In both, the dementia praecox is complicated by convulsions, which the author considers as symptomatic convulsions in catatonia.

We avoid much difficulty if we consider the convulsion independently of any disease concept. We must look on the convulsion merely as one of the many types of motor disturbance occurring in catatonia, and consider this type of case as a peculiar constitutional coloring of the catatonia. As we have symptomatic convulsions in other diseases, so we have them here, and we are not justified in assuming a combination of two diseases.

SELLING, Portland, Ore.

STUDIES ON THE VISCERAL SENSORY NERVOUS SYSTEM. XIV.
THE REFLEX CONTROL OF THE CARDIA AND LOWER
ESOPHAGUS IN MAMMALS. A. J. CARLSON, T. E. BOYD and J. F.
PEARCY, Arch. Int. Med. **30**:409 (Oct.) 1922.

The authors point out that a knowledge of the reflex control of the cardia and lower esophagus is important in explaining spasms of the cardia and esophagus in man. The animals used in the experiments were cats and dogs. One dog was provided with a permanent gastrostomy and an esophageal fistula in the neck, and was used for the study of reflexes to the cardia from the mouth, pharynx and stomach, the action of cocaine on the cardia, etc., without anesthesia. In the acute experiments,* the animals were anesthetized with ether. Gastrostomy and fistula of the esophagus were made for fixing a cardiometer in place, and usually a balloon was also placed in the stomach for recording the gastric tonus. The procedure after this was as follows: (1) conduction of experiments under constant, but light ether anesthesia; (2) ether anesthesia continued with curare and artificial respiration, for elimination of skeletal and respiratory reflexes; (3) animal decerebrated and ether anesthesia discontinued; (4) the foregoing conditions followed with chest and respiratory mechanism intact, or with chest opened for artificial respiration, the phrenic nerve sectioned and the diaphragm dissected away from the esophagus to eliminate possible errors from spasm of the diaphragm.

In normal dogs, not under anesthesia, the tonus of the cardia was temporarily inhibited by stimulation of sensory nerves in the mouth and pharynx and by stimulation of nerves in the gastric mucosa; it was increased by sudden distention of the walls of the stomach, by intravenous injections of small quantities of cocaine, by some factor other than the acidity of the gastric contents during gastric digestion. The tonus of the cardia was found to run parallel with the tonus and hunger contractions of the empty stomach.

In dogs and cats under light ether anesthesia, ether and curare or decerebration, it was found that reflex inhibition or contraction of the cardia and

lower esophagus can be initiated by the stimulation of any sensory nerve skeletal or visceral; that when the vagi are intact, stimulation of the sensory nerves in the mouth, pharynx, esophageal and gastric mucosa induce on the whole inhibition of the cardia, followed by contraction; that stimulation of the afferents from the abdominal viscera (gallbladder, intestine, urinary bladder, central end of one of the splanchnic nerves) induces, on the whole, reflex contraction of the cardia and lower part of the esophagus, even when both vagi nerves are sectioned; that when the tonus of the cardia is feeble, the motor reflexes into the cardia predominate, and when the cardia is hypertonic, the inhibitory reflexes prevail; that strong stimulation of the abdominal viscera of the central end of the splanchnic nerve may cause strong spasm of the cardia and lower esophagus lasting from ten to thirty minutes.

The authors note the fact that the tonus of the cardia being inhibited reflexly by chemical and mechanical stimulation of the sensory nerves in the mouth may have some practical application. Bitters or acids in the mouth may reflexly help to inhibit the milder forms of reflex cardiospasm.

VONDERAHE, Philadelphia.

LOCALIZATION OF APRAXIA. FORSTER, *Monatschr. f. Psychiat. u. Neurol.* 50:1 (July) 1921.

In 1912, the author reported a case of bilateral apraxia with associated symptoms pointing to the left-hand center (right hemiparesis, jacksonian attacks beginning in the right hand). Operation revealed a glioma lying just beneath the surface at the base of the second frontal convolution. The findings were confirmed by postmortem examination. In presenting the case at the time, the author assumed that the apractic symptoms were referable to the frontal lobe lesion. The possibility of their being the result of a lesion of the precentral gyrus could not be excluded.

Subsequent study of the case with serial sections demonstrated that there were three tumors: one, already described, involved the foot of the second frontal convolution; a second lay in the substance of the first frontal convolution, a little posterior to this; the third involved the anterior portion of the corpus callosum.

In his original presentation of the case, the author said he believed that it proved frontal lobe lesions capable of causing apraxia. His subsequent studies failed to bear out this contention. Areas of degeneration were found in the central convolution so that certain connections between frontal lobe and central convolution must have been lost. Furthermore, the central convolutions were cut off from the corresponding areas of the right hemisphere by the callosal tumor. There was no obvious disturbance in the connections between the central convolutions on the one hand, and the occipital and temporal convolutions on the other. But the existence of areas of rarefaction in the subcortical zone of the central convolutions compels the admission that such breaks may have existed.

Kleist, as a result of the study of war material, believes that limb-kinetic apraxia results from involvement of the precentral gyrus, whereas ideokinetic apraxia results from left-sided parietal lobe lesions. Left-sided apraxia in right-sided paralysis occurs (with one exception) only when the paralysis is accompanied by sensory disturbances and touch agnosia. Kleist concludes that the impulses regulating the functioning of the left arm reach it, not through

the left precentral gyrus, but from the left parietal zone. If the parietal zone is undamaged, there must be involvement of the callosal fibers coming from the depths of the parietal lobe, or of the corpus callosum itself.

In the author's case, neither parietal lobe nor deep callosal fibers, nor the posterior portion of the corpus callosum were involved. But the tumor of the anterior portion of the corpus callosum may have thrown the posterior portion out of function by pressure.

The bilateral apraxia in this case may be explained as due to lesion of the left precentral gyrus; and the left-sided apraxia is explainable in addition on the basis of damage to the corpus callosum in this region. The assumption that the parietal lobe either through pressure or through injury to its callosal fibers may have in part been responsible for the loss of function cannot be entirely excluded.

SELLING, Portland, Ore.

THE PALM-CHIN REFLEX. A. RADOVICI, *Ann. de méd.* **12**:56 (July) 1922.

The author points out that the cutaneous reflectivity of the upper extremity is less than that of the inferior extremity. This difference finds its explanation in the fact that movements of the upper extremity are eminently voluntary, since the arm serves to bring about movements of precision toward a determined end. In contrast, maintenance of the erect posture and the automatism of walking are accomplishments pertaining to the lower extremity, and carry with them the greater development of reflexogenic phenomena. The skin of the thorax, arms, forearms and palmar surface of the hand are classically considered as exempt from cutaneous reflexes. However, two years ago, in collaboration with Marinesco, the author described the existence of a palmar cutaneous reflex. The excitation of the palm of the hand produces a contraction of the muscles of the chin on the same side. This so-called palm-chin reflex exists in about 60 per cent. of normal persons. Normally, there is often a difference between the right and left reflex in favor of the left. It is suggested that the right arm is more strongly differentiated from the view of voluntary motility and therefore presents less reflex activity than the left.

The palm-chin reflex is produced by a rapid stroking of the thenar eminence with a sharp point, and consists of a quick contraction of the muscles of the chin on the same side, producing a marked lifting of the lower lip. There must be preliminary relaxation of the lip and the mouth must be half open.

The examination of the palm-chin reflex gives indications for the diagnosis of the site of lesions of the pyramidal tracts in their superior portions. The unilateral exaggeration of the reflex indicates a bilateral supranuclear lesion of a pyramid above the seventh nerve nucleus. A bilateral exaggeration of the reflexes indicates a bilateral supranuclear lesion of the pyramids: pseudobulbar paralysis, amyotrophic lateral sclerosis and Little's disease. A nuclear or peripheral lesion of the facial nerve abolishes the reflex on the side of the lesion. A high cervical spinal lesion above the brachial swelling can bring about a modification of the reflex. In one case the palm-chin reflex was not present, but excitation with a needle of the palm or thenar eminence produced an extension of the thumb (analogous to the Babinski sign).

A total lesion at the brachial swelling abolishes the reflex, while a partial lesion, which does not affect the eighth cervical and first dorsal segments can

produce a modification of the reflex, so that palmar excitation causes muscular contraction of the pectorals, supplied by segments which are unaffected.

The existence of the reflex in high dorsal lesions proves that the lesion is inferior to the first dorsal, the lowest brachial segment.

DAVIS, New York.

RESULTS OF PUNCTURE OF THE CORPUS CALLOSUM. POHLISCH, *Monatschr. f. Psychiat. u. Neurol.* **50:251** (Nov.) 1921.

The author gives a summary of the results of callosal puncture, introduced by Anton and von Bramann, in 1908. The operation was recommended by its authors as a relatively simple method of procuring permanent ventricular drainage, applicable as a palliative measure to practically all conditions of intracranial pressure increase. Pohlisch reports ten cases of his own, and has reviewed 550 cases from the literature. He compares the method and its results with ventricular puncture and decompression. Spinal puncture is not considered because of the dangers attendant on its use in case of pressure increase.

Operative Procedure: Callosal puncture is simpler than decompression, but no simpler than ventricular puncture. There are definite possibilities of trouble in the form of hemorrhage, thrombosis and collapse resulting from too rapid pressure reduction. In view of these complications, the author considers the procedure hardly less serious than ventricular puncture or decompression.

Diagnostic Significance: Occasionally one can palpate with the cannula a tumor lying in the wall of the ventricular cavity. Aside from this, callosal puncture offers no advantages over ventricular puncture or decompression.

Therapeutic Results: (a) Hydrocephalus. Perhaps one third of all cases are favorably influenced. Focal symptoms show little improvement, but the general pressure symptoms (headache, dulness, vomiting, vertigo, convulsions), and especially the choked disk, may be considerably improved. In a number of cases it was possible to save vision. In a few cases more permanent results were obtained. The question of permanency of results is bound up with the question as to whether the drainage canal through the corpus callosum remains open. A small number of cases have been studied at necropsy. In one, the puncture was found closed after seven days; in another, it was open after two and one-half years. The author's conclusion is that in hydrocephalus, callosal puncture has no advantage over single or repeated ventricular puncture. (b) Brain tumors. In this group, as in the preceding, callosal puncture has no advantage over ventricular puncture. (c) Epilepsy, tower skull, migraine. Anton and von Bramann suggested the use of callosal puncture in these groups of cases, but there is nothing to indicate that it is of any value. (d) Serous meningitis. According to Paye, callosal puncture has been of value in the treatment of cases of traumatic serous meningitis. His records did not indicate whether the benefit was due to the immediate pressure relief or to permanent drainage. Others have obtained similar results in the same type of case by ventricular puncture, and even by spinal puncture.

SELLING, Portland, Ore.

THE BASAL METABOLISM IN NONTOXIC GOITER AND IN BORDERLINE THYROID CASES, WITH PARTICULAR REFERENCE TO ITS BEARING IN DIFFERENTIAL DIAGNOSIS. JAMES H. MEANS and HARRIET BURGESS, *Arch. Int. Med.* **30:507** (Oct.) 1922.

The authors analyze the data presented by basal metabolism determinations on 1,000 patients, with reference to the diagnosis of thyroid activity.

Five groups of cases are discussed in this paper. Cases showing subnormal metabolism, lower than minus 10 per cent., represent the first group. Low basal metabolism occurred in all untreated patients, numbering twenty-six, with myxedema and cretinism. Sixty-three others presented a low metabolic rate; 84 per cent. of these either had endocrine disorders or presented some clinical suggestion of endocrine disease; in the remaining, the subnormality in rate was slight. The endocrine diseases other than thyroid included suprarenal and pituitary abnormalities, and a probable case of Addison's disease. In anemia, polycythemia and leukemia, subnormal metabolism was found to be a distinct rarity. In the second group are placed cases with a supernormal metabolism. Of 300 cases showing clinical signs and symptoms of hyperthyroidism, only one failed to show a metabolic rate of plus 10 per cent.; this patient had mild symptoms. Supernormal metabolism was found in blood diseases, in leukemia and severe anemia and in 24.4 per cent. of endocrine disorders other than hyperthyroidism. Twenty cases, nonendocrine in character, presented increases in metabolism; these included a woman seven months pregnant, patients with malignant lymphoma of the neck, osteitis deformans, paroxysmal tachycardia and probable chlorosis. In a case of paralysis agitans, the first determination was plus 23 per cent.; the second determination made a week later when the patient was more quiet was minus 2 per cent. In the third group are 102 cases of nontoxic goiter. All of these cases gave metabolic rates within normal limits. A fourth group is made up of seventy patients suspected of having hypothyroidism, most of whom had normal metabolic rates; those that had subnormal rates improved under the administration of thyroid extract. A fifth group was made up of 290 patients suspected of having hyperthyroidism; two thirds of these gave metabolism rates with normal range, one third were supernormal. Errors, the authors note, are always in the direction of higher readings, so that a normal rate in the presence of a suspected hyperthyroidism rules out this condition.

On the basis of the evidence presented the authors conclude that, provided certain well recognized causes for increased metabolism, such as fever, acromegaly, leukemia and severe anemia, are excluded, the finding of an increased metabolic rate is strong presumptive evidence of hyperthyroidism; and that, provided causes for decreased metabolism, such as starvation, hypopituitarism and hyposuprarenalism, are excluded, a low metabolic rate is strong presumptive evidence of hypothyroidism.

VONDERAHE, Philadelphia.

TETANY. A REPORT OF CASES WITH ACID-BASE DISTURBANCE. SAMUEL B. GRANT, Arch. Int. Med. **30**:355 (Sept.) 1922.

The cases of tetany reported occurred in adults. In all instances studied there was a marked increase in the sodium bicarbonate of the blood plasma. Three of the cases occurred in obstruction of the stomach; in one of these cases the blood chlorid was markedly decreased, but the blood calcium was within normal limits. Two cases occurred following overdosage with sodium bicarbonate. In one of these cases the soda was given in the course of a Sippy treatment for gastric ulcer. On the fifth day a prickling sensation of the hands developed, followed later by a flexion spasm of the fingers; when alkali was discontinued no further spasm occurred. The plasma carbon dioxid capacity the following morning was 90.5 volume per cent., and at this time there was a positive Trousseau sign, flexion spasm of the fingers, adduction of the thumb and a faint Chvostek sign. When the treatment was continued

with smaller doses of alkali bringing the plasma carbon dioxid capacity to 71 volume per cent., the signs of tetany disappeared. In the other case sodium bicarbonate was given intravenously and by Murphy drip in a case of severe acidosis accompanying toxemia of pregnancy. A rise of the carbon dioxid capacity from 19.5 volume per cent. to 27 volume per cent. appeared. Sodium bicarbonate, 2 gm. every two hours, was given by mouth in addition. Three days after admission the carbon dioxid capacity was 67.5 per cent., and the following day the patient developed carpal spasm with twitching about the mouth. At this time the carbon dioxid capacity had risen to 87 volume per cent.

The last case reported accompanied hysteria in a nurse, who, failing in her examinations, developed an attack characterized by deep and rapid breathing, after which she complained of numbness and tingling in her fingers. After stopping the hyperpnea, the sensations disappeared. Later the rapid, deep breathing returned, followed by tingling and numbness and later by flexion spasm of both hands. When she was persuaded to breathe quietly, the spasm subsided. Trousseau's sign was positive. Unfortunately, the carbon dioxid capacity of the plasma was not determined; the explanation suggested is that the hyperpnea "washed out" the carbon dioxid from the blood, leaving a relative excess of sodium bicarbonate.

VONDERAHE, Philadelphia.

CONTRIBUTION TO OUR KNOWLEDGE OF LESIONS OF THE
SUBTHALAMIC REGION. BRESOWSKY, Monatschr. f. Psychiat. u.
Neurol. 50:302 (Nov.) 1921.

A patient, aged 39, had an attack of influenza followed by a right hemiplegia and mental symptoms. Neurologic examination, except for the usual findings in hemiplegia, was negative. One month later left-sided ptosis developed, eye muscle involvement and hemianopsia, all transient in character. Ten days later there was definite protrusion of the right eyeball. This unilateral exophthalmos gradually increased in degree. There were repeated convulsions. Death came suddenly, two and a half months after admission.

Necropsy revealed an encapsulated abscess of the left thalamus, which extended downward on the same side, destroying the red nucleus and part of the substantia nigra, and rupturing caudal to the corpus subthalamicus. Practically the whole thalamus, excepting the ventro-caudal and ventro-medial portion in the neighborhood of the corpus subthalamicus, was destroyed. The corpus subthalamicus itself was not involved.

The chief interest in the case centers in the author's interpretation of the right-sided exophthalmos. He discards the idea that it might be a distant symptom. He looks on it as a strictly focal symptom, due to irritation of the subthalamic area. It developed one month before exitus, at a time when, according to the history, the abscess was sinking from the thalamus toward the subthalamic region. This was indicated by the development of midbrain symptoms.

The author points out that for a long time the central gray of the mid-brain has been associated with the central origin of the sympathetic system. Karplus and Kreidl demonstrated in experimental animals that stimulation of a certain point in the subthalamic region would produce the same symptoms as stimulation of the cervical sympathetic: dilation of the pupil, widening of the lid slit, and exophthalmos. In their experiments, the symptoms were chiefly contralateral. In the author's case, the exophthalmos was also contra-

lateral. The same was true of a similar case reported by Schrottenbach. Gerstman has reported one with homolateral symptoms. According to the author's case, the crossing of the fibers must be caudal to the subthalamic region, not oral as Gerstman believed.

SELLING, Portland, Ore.

A PRELIMINARY STUDY OF THE PRECIPITATING SITUATION
IN TWO HUNDRED CASES OF MENTAL DISEASE. EDWARD A.
STRECKER, *Am. J. Psychiat.* **1**:503 (April) 1922.

This is a study of a fundamental psychiatric problem, that of the "precipitating situation," and incidentally of interest to those who in compensation, military and legal cases have to give reasons for their belief that a given incident is or is not a cause of a following psychosis.

In 100 cases each of dementia praecox and manic-depressive psychoses, Strecker gives in parallel columns a condensation of the psychic and physical situation at the onset of the disease and an opinion as to the importance of the special situation as a precipitant of the attack. Of 200 patients, 107 combined psychic and organic factors, forty apparently followed somatic disturbances alone, thirty-nine followed psychic disturbances, and fourteen failed to reveal any reason for the breakdown.

Apparently fully adequate precursors of breakdown appeared in thirty-two manic-depressive and in twenty dementia praecox patients, while no stress appeared before the onset in only two manic-depressive and in twelve dementia praecox patients. In both illnesses there was some evidence that the emotional tone proper to the important prepsychotic situation was carried over into the psychosis itself. It was suggested also that the accumulation of mental and physical strain over a considerable period of time may exceed in importance a single dramatic event. Mental diseases in the family and abnormal personalities appeared most frequently when the precipitating situation seemed trivial. In manic-depressive cases recurrences were also found most often when the original unfavorable elements in the situation were doubtful, insignificant or absent.

All careful clinical work in this direction, and even careful speculation, is valuable. We have come to a time when the study of physical aspects of a psychosis need not turn us away from its mental aspects. In the situations in this paper, how can we be sure that "loss of weight" is not a different side of "an unhappy marriage," or separate the worries about illegitimate pregnancy from the exhaustion of childbirth? So that the crux of Dr. Strecker's paper is not the question of mental against physical, but the question, "Of what importance was any particular set of factors in the onset?"

BOND, Philadelphia.

EXPERIMENTAL DIABETES INSIPIDUS. PERCIVAL BAILEY and FRÉDÉRIC BREMER, *Arch. Int. Med.* **28**:773 (Dec.) 1921.

This work describes systematic punctures of the hypothalamus in dogs, with observations on the symptoms following the operations. In operative study of the hypophysis this area is liable to injury, so that a study of its function becomes important in interpreting the results of previous experimentation.

The hypothalamus was explored by means of the lateral route; in each instance the hypophysis was left intact. Puncture of the parainfundibular

region of the hypothalamus in every one of the thirteen dogs produced polyuria which appeared within the first two days following the operation; in extensive lesions the polyuria became permanent.

The polyuria induced in the dogs was such that concentration of urine occurred when fluid intake was restricted or when the pituitary extract was injected subcutaneously or in the presence of fever, while excessive polyuric action was noted after the administration of chloride; the theobromin effect was absent. These reactions, the authors note, are found in diabetes insipidus of man. Thirst, with an increased intake, often appeared before the polyuria, but was not constant; in comatose animals polyuria appeared without intake of water. In animals whose kidneys had been denervated the polyuria persisted without essential change. In two cases in which the tuber cinereum was injured, a cachexia "hypophyseopriva" with genital atrophy developed; in two other similar cases with injury to the tuber cinereum there was an insidious development of adiposogenital dystrophy; in every instance there was a persistent polyuria. Animals with an extensive lesion of the tuber cinereum either died in a short time or after a period of coma and convulsions. Glycosuria was inconstant in its appearance.

The authors perceive clearly that this work questions the reliability of previous studies leading to the widely accepted opinions that adiposogenital dystrophy is due to a deficiency in the secretion of the anterior lobe of the pituitary and polyuria to a deficiency of the posterior lobe secretion. For this previous teaching they substitute the tuber cinereum as being concerned with the syndrome of adiposogenital dystrophy and the hypothalamus with polyuria. In the discussion of their observations they defend this view with considerable vigor and answer the objections which may be raised against it.

VONDERAHE, Philadelphia.

HEMIPLEGIA WITH HEMICHOREA. A. REMOND and H. COLOMBIES, Rev. de med. **39**:107 (Feb.) 1922.

This is a clinical and necropsy report of a case showing combined hemiplegia and hemichorea. A woman, aged 70, was suddenly aware of an intense tingling of the entire left side of her body; she then rapidly lapsed into unconsciousness and became hemiplegic (left-sided). At the moment that consciousness was lost, irregular disordered movements of moderate intensity began in the left arm. The face was animated by continuous spontaneous movements predominating on the left. Both the coma and the choreiform movements remained constantly until death after three days. The left arm, flaccid at the shoulder, showed a marked tonic of the forearm. The left leg, though flaccid, showed a positive Babinski sign. This sign was not present on the right. The tendon reflexes were not obtained on either side. The extremities of the right side presented normal tonus. The pupils were myopic and gave no reaction to light. The spinal fluid was transparent, not under pressure, and was not abnormal on examination.

Necropsy revealed a cerebral hemorrhage "the size of a large nut," situated just under the cortex in the superior portion of the perirolandic area. It was sharply limited, did not inundate the ventricles and did not attain any of the gray nuclei. Additional sectioning of the brain did not reveal any other lesions either recent or cicatrized.

The authors point out that such a hemiplegic and hemichoreic association is rare, and that incertitude holds regarding the lesion capable of producing it. They consider, however, that their case correlates with the ideas of Pick

and Kahler, who incriminate "an irritation of pyramidal tract fibers or of motor centers." The perirolandic cortex, caught between the cranial vault and the hemorrhage mass, received a constant irritation, made evident by motor reactions. They are carried only by the fibers not sectioned by the lesion, in their case, the fibers supplying the face and arm.

Touche has written that in almost all cases of hemiplegia with hemichorea there are multiple lesions. But the case reported had only one lesion, sharply delimited.

DAVIS, New York.

EXPERIMENTS ON THE DEVELOPMENT OF THE CRANIAL GANGLIA AND THE LATERAL LINE SENSE ORGANS IN AMBLYSTOMA PUNCTATUM. L. S. STONE, *J. Exper. Zool.* **35**:421. (May 20) 1922.

The experiments were for the purpose of extending knowledge concerning the part which placodes and neural crest play in the formation of cranial ganglions and nerves and also to determine the extent of their contribution to the formation of the mesoderm and the exact origin and fate of the "mesectoderm" tissue. Above the optic vesicle in early stages there is an elongated ophthalmic placode which gives off cells to the formation of the ophthalmic ganglion. Near the anteroventral border of the supra-orbital lateral line primordium is a small gasserian placode of brief duration. Close to the anterior border of the auditory placode is a prominent placode elongated toward the dorsal extremity of the hyomandibular cleft. Removal of this gives rise to the absence of the supra-orbital line of sense organs and a large part of the seventh lateral-line ganglion. The supra-orbital primordium of lateral-line sense organs arises from the anterior extremity of the seventh lateral-line ganglion placode. The supra-orbital, infra-orbital and hyomandibular primordia of lateral-line sense organs have separate seats of origin. The ventral hyomandibular and mandibular groups of lateral-line organs appear to have separate seats of origin. The epibranchial placodes of the seventh, ninth and tenth segment give off cells which become incorporated in the visceral ganglions. Removal of ectoderm in the region of the ninth and tenth segments results in a complete absence of lateral-line ganglions. Cutaneous as well as visceral sensory fibers are absent. The neural crest cells, from the dorsal portion of the neural tube, can be distinguished as early as the closure of the neural folds. The wandering mass of "mesectoderm" is of neural crest origin and is not augmented by a contribution from cells of the lateral ectoderm. The hyohyals, ceratohyals, ceratobranchials, epibranchials and first basibranchial are formed from the wandering neural crest in the branchial and hyoid regions. In the trigeminal region the neural crest cells form the quadrate and mandibular cartilages. The neural crest in the trigeminal region which migrates over the anterior border of the optic vesicles apparently gives rise to the anterior portion of the trabeculae.

WYMAN, Boston.

CLINICAL STUDIES ON THE CAUSE OF SCHIZOPHRENIA. II. ERWIN POPPER, *Monatschr. f. Psychiat. u. Neurol.* **50**:235 (Oct.) 1921.

In the foregoing paper the author discussed the occurrence and significance of endogenic factors in the development of schizophrenias. He points out that endogenic factors, as represented by heredity, disposition and prepsychotic personality, are more or less vague in character and difficult to evaluate.

Nevertheless, the frequency with which they appear in the histories of schizophrenias is such that we must recognize their importance in the development of the disease. In one sixth of all cases in this series, no endogenic factors could be determined, and it is this group particularly that forces us to a consideration of exogenic factors. Here, again, we encounter difficulties. A mere time relationship between a trauma or shock and the development of the disease is not sufficient; we must demonstrate that some more intimate connection exists. A further difficulty lies in the fact that not every psychosis which resembles schizophrenia actually belongs to that disease group. It is probable that some of the cases in which exogenic factors play an especially prominent rôle are not true schizophrenias, but reactive psychoses of different types. Summarizing his studies, the author concludes that we must assign to endogenic factors a primary significance in the development of schizophrenias. But considering the fact that one sixth of all cases show no endogenic factors, together with the frequency and intimacy with which exogenic injuries of one kind or another are associated with the outbreak of the disease or one of its episodes, we must admit that exogenic factors do have an etiologic significance.

SELLING, Portland, Ore.

TUBEROSE SCLEROSIS AND HYDROCEPHALUS AND THEIR ASSOCIATION WITH PRECOCIOUS PUBERTY. K. H. KRABBE, *L'Encéphale* 17: No. 5, pp. 281-289; No. 7, pp. 437-445; No. 8, pp. 496-506.

The etiology and pathology of premature sexual development are not clearly understood. In this paper, Krabbe discusses precocious puberty and its association with cerebral affections and, in particular, certain forms of hydrocephalus, tuberoses sclerosis and pineal tumors. Precocious puberty due to tumors of ovarian or testicular origin forms a group of cases not germane to the author's study.

Numerous cases of several types are presented in detail, and there is a brief treatment of the literature bearing on these cases.

Krabbe points out that the presence of precocious puberty in certain conditions is no proof that it is caused by them. In cases of pineal tumor, necropsies have too frequently been limited to the head. In Krabbe's experience with pineal tumors, pathologic changes may be found elsewhere in the body, particularly in the endocrines. Moreover, in pineal tumors other things than the destruction of the pineal gland must be considered as possible factors in the production of precocious puberty—secretion of particular substances of the tumor involving the gland, action à distance on the pituitary and action on the cerebral sympathetic centers about the third ventricle.

Both from his own cases and from the literature Krabbe concludes that these local brain diseases are evidence of a general malfunction or disorganization of certain groups of tissues and organs and that precocious puberty is also evidence of this same anomaly. However, it may depend not only on the underlying anomaly, but on the presence of one or more other associated factors, such as pineal destruction or disturbed function in the cerebral portion of the sympathetic system. Krabbe emphasizes that any of the three of the cerebral conditions with which his paper deals may be present without distinct premature sexual development. This is particularly true in cases of pineal tumor and hydrocephalus. In fact, he has observed that hydrocephalus, as well as tumor, or destruction of the pineal gland, may coexist without consequent precocious puberty.

HYSLOP, New York.

TRANSITORY AGNOSIA OF AN INFANT IN REGARD TO ITS ARMS.

AUGUSTE TOURNAY, *Rev. neurol.* **29**:580 (May 4) 1922.

This report gives detailed daily observations on the lack of recognition of its hands (agnosia) by an infant from the age of 115 days to that of 141 days, as well as daily observations on the disappearance of the Babinski sign of infancy. The relation of these observations to the anosognosia of certain persons with left hemiplegia, described in 1914 and 1918 by Babinski, is discussed.

It was noticed that the child at the one hundred and fifteenth day began to take definite notice of its right hand, while it paid absolutely no attention to its left. The child was and is quite normal in every way, and is now 2 years and 4 months old. Vision in all fields was and is normal. This agnosia, involving the left hand and not the right, continued for about twenty-six days, after which the child began to become interested in and to take notice of its left hand. From that time on it was equally interested in both. Therefore for 115 days of its life it had no apparent power to appreciate itself (hands) or to differentiate its hands from the outside world. At this time it became conscious of its right hand as belonging to itself. After a period of twenty-six days, it became conscious of its left as belonging to itself.

One hundred and eighty-one days after birth, stimulation of the feet for the first time showed flexion of the great toe on the right, but on the left there was still extension. This did not change to flexion until eleven days later.

Tournay relates these findings to the general question of agnosia, as well as to the anosognosia of Babinski—the failure of some persons with left hemiplegia with associated sensory disorders to pay any attention to their paralyzed arm.

He also emphasizes the asymmetrical disappearance in his case of both a sensory (agnosia) and a motor (Babinski sign) phenomenon, and the fact that these first disappeared on the same side—the right.

KRAUS, New York.

NEURORECURRENCES FOLLOWING TREATMENT WITH ARSPHEN-

AMIN. ERNEST L. ZIMMERMANN, *Arch. Dermat. & Syph.* **5**:723 (July) 1922.

The author studied thirty-nine cases of primary and early secondary syphilis in which neurorecurrences developed during the course of treatment with arsphenamin. This number represents an incidence of 1.64 per cent. In every instance there was a lapse in the treatment, usually from four to eight weeks before the development of a neurorecurrence.

The symptoms of a neurorecurrence are divided into three groups: (1) acute syphilitic meningitis, with or without focal lesions; (2) meningitis of moderate or slight intensity, manifesting itself chiefly by headaches and focal lesions, and (3) no general symptoms of meningitis, the condition being due to a focal lesion. The facial and auditory nerves were found to be involved most frequently.

The author accepts Ehrlich's explanation of neurorecurrence, based on the theory of a sterilization which just falls short of being complete, the host failing to develop resistance against the spirochetes which survive in isolated foci. The pathology is based by the writer on the study of P. Pirilla, who found a diffuse meningovascular process, and on the study of J. H. Lloyd,

who found a localized lymphocytic infiltration. Clinically, the spinal fluid is abnormal in the former; in the latter, it may show marked abnormalities or may be entirely normal.

Only two of the patients had used inunctions to a considerable extent; no patient who had persisted in the course of mercury following arsphenamin developed precocious neurosyphilis. The prevention of neurosyphilis is thus dependent on thorough mercurial treatment.

VONDERAHE, Philadelphia.

AN UNUSUAL CALCIFICATION IN THE BRAIN. WEIMANN, *Monatschr. f. Psychiat. u. Neurol.* **50**:202 (Oct.) 1921.

The author gives a detailed study of a case of so-called "calcification of intracerebral vessels." A few cases are reported in the literature, all with the same essential features. Throughout the brain, involving both hemispheres and reaching as far down as the pons, is a widespread deposit of calcium salts. The primary deposit appears in the perivascular lymph spaces. From here it gradually invades the vessel wall, involving first the adventitia, then the media, and finally the intima. When the process is extreme, the whole vessel is converted into a rigid tube, which, on section, stands out above the surrounding tissues. The calcium appears as small concretions or as a finely granular deposit, and if many adjacent capillaries are involved, the whole parenchyma may be filled with coalescing calcium masses. Microchemical studies show that the calcium is deposited in a colloidal (protein) groundwork. The picture differs entirely from that of a generalized arteriosclerosis. The sites of election are the centrum semiovale, the basal ganglions and the cerebellum. In none of the reported cases has the process extended below the level of the medulla.

Calcification of the intracerebral vessels is a secondary process. It has been observed in various types of local and diffuse degeneration of brain tissue, congenital and acquired idiocy, epilepsy, general paresis, focal softening, etc. In almost all cases, the findings suggest a marked disturbance of lymph circulation. This is probably the primary factor determining the deposition of the calcium salts. Secondary factors depend on the chemistry of blood and tissue fluids.

The clinical picture is not characteristic. It depends on the essential nature of the degenerative process and varies in the different types of cases.

SELLING, Portland, Ore.

PITUITRIN AS A MUSCULAR TONIC. AN ERGOGRAPHIC STUDY. URECHIA and GRAFF, *Ann. de med.* **12**:64 (July) 1922.

Struck by the influence of pituitrin on the asthenia of diabetes insipidus, the authors made more ample investigations regarding its action, and have carried out experiments using patients with optostriate or optopeduncular lesions. In epidemic encephalitis, in paralysis agitans and in diabetes insipidus the asthenia is a disturbance of tonus, and in all of these affections neither the suprarenal nor hypophysis presents important lesions which can explain the asthenia.

They employed pituitrin, injecting it in doses varying from 0.01 to 0.02 or 0.03 gm., making ergographic and kymographic curves in thirteen patients before the patients had received pituitrin injections in series as medication, and after such series. The curves were made every half hour during the four hours following an injection. They left intervals, some as long as twenty-three days,

between repetitions of the test in the same patient in order to avoid the misleading effects that "training" might produce. Their tabulations give the number of movements of the weight before exhaustion, and the number per minute, also the distance and the work (weight times the distance), the time before exhaustion and the force (namely the weight times the distance divided by the time). In almost all instances, the curves obtained showed an increasing work and force, presumably due to the injections.

Pituitrin also showed incidentally a marked affect on hypersomnia, especially in one case. This patient, who went to sleep in his chair in the laboratory, could not sleep after the pituitrin injection, sometimes for three to four hours, sometimes for a day. When the injections were stopped, the hypersomnia reappeared, and then disappeared anew when pituitrin was resumed. But it is fortunately only on pathologic somnolence that pituitrin has an influence.

DAVIS, New York.

THE FUNDAMENTAL PLAN OF THE VERTEBRATE BRAIN. B. F. KINGSBURY, *Jour. Comp. Neurol.* **34**:461, 1922.

The longitudinal zones of the early neural tube as defined by His are reexamined in their relations with the plan of the neural plate in still younger stages and with the morphology of the head as a whole. The neural plate ends anteriorly at the chiasma ridge (Johnston) and the primordia on the neural plate of the two optic vesicles are connected across the median plane by a primitive optic furrow just spinalward of this ridge. The anterior end of the floorplate of His is at the fovea isthmi immediately dorsally to the rostral end of the notochord. In front of this boundary the two basal plates fuse in the median plane as far forward as the mammillary recess. Still further forward the two alar plates fuse medially as far forward as the preoptic recess. These alar plates undergo precocious development anteriorly in higher vertebrates and give rise to the whole of the cerebral hemispheres. Their secondary fusion rostrally of the chiasma ridge closes the anterior neuropore, thus forming the lamina terminalis.

These conclusions agree in the main with the well-known views of Johnston and require considerable revision of other current conceptions of the relations of the zones of His in the forebrain. The sulcus limitans is believed to end in the mammillary recess, no part of the primitive motor zones of the neural tube lying rostrally to this region.

C. J. HERRICK, Chicago.

RECOVERY FROM TUBERCULOUS MENINGITIS. A. CRAMER and G. BICKEL, *Ann. de méd.* **12**:226 (Sept.) 1922.

Tuberculous meningitis is generally regarded as fatal, and recovery is so rare that the accuracy of the diagnosis is always questioned.

In this paper, the authors perform a distinct service. They have collected reports of forty-six cases of tuberculous meningitis in which the patient recovered and in which the diagnosis could not be questioned, and from this material an attempt to determine the conditions permitting recovery is made.

Tuberculous meningitis is almost always a complication of tuberculous infection elsewhere in the body. In early life, before the body defenses against tuberculosis have had an opportunity to develop, tuberculous meningitis is almost invariably fatal. The authors found that only one patient under 2 years of age recovered and only six between the ages of 2 and 5. Thus, of

all who recovered, only 17.5 per cent. were less than 5 years old. (When we consider that the great majority of cases of tuberculous meningitis occur in early life and that in children under 12 years of age 83 per cent. of the cases develop before the fifth year—the factor of individual resistance to the infection is of great importance.)

Of the other patients who recovered, 25 per cent. were from 5 to 10 years of age, 2.5 per cent. from 10 to 20 years and 30 per cent. over 20 years.

Among other factors favoring recovery are absence of hypertension of the spinal fluid and low virulence of the causative organisms.

Several methods of treatment reported as contributing toward recovery are mentioned by the authors, but it is their belief that the reason for recovery lies in the resistance of the patient.

Finally, it must not be forgotten that in about 25 per cent. of reported recoveries, there was, in time, a fatal recurrence of the infection.

HYSLOP, New York.

STUDIES IN TRAUMATIC FRACTURES OF THE CRANIAL BONES:

I. EDEMA OF THE BRAIN. II. BRUISES OF THE BRAIN. C. W. APFELBACH, Arch. Surg. 4:434 (March) 1922.

The author notes that cerebral edema has not received the attention given to other sequelae following fracture of the cranial bones. The water content of twenty-six brains removed from persons who died of linear fractures of the cranial bones and associated brain injuries was determined, using the method devised by Waldemar Koch and modified by Mathilde Koch. The water content of six control brains was also studied. The actual increase of water for the entire brain when edema was present varied between 3 and 45 gm.

In the twenty-six cases of the author a division of the bruises of the brain was made into two groups: those which result from laceration of the brain tissue at the time the cranial bones are fractured, and those caused by hemorrhage from torn pial arteries at or near the junction of the gray and white matter. The latter group is made the object of particular study. The brains were examined after the leptomeninges were removed. The bleeding was noted as taking its origin at the junction of the gray and white matter and proceeding to the outside of the brain. The author suggests that gray matter, being more cellular, favors the spread of bleeding.

VONDERAHE, Philadelphia.

THE CASTES OF TERMOPSIS. CAROLINE BURLING THOMPSON, J. Morphol. 36:495 (Sept. 20) 1922.

The two species *angusticollis* and *nevadensis* of the genus *Termopsis* are found on the Pacific slope and in the northwestern United States. The habitat is the decaying wood of forests, rarely in buildings, and never in the earth. Four stable castes are of common occurrence; the first, second and third forms are the fertile reproductive castes; among the soldiers the females are sterile; the males are probably also sterile, though near fertility. There is no true sterile worker class. The sexes are differentiated externally in all castes. Three additional types are of occasional occurrence, second and third forms with small wing vestiges and soldiers with wing vestiges. There is no corre-

lation between the presence of wing vestiges and fertility, but brain size and fertility are invariably correlated. A platelike, nonfunctional frontal gland, without fontanel, is present in first-form nymphs and adults. Vestiges of the lateral ocelli are found in all the castes, except, possibly in the third form. There is a great variability in all organs, and even in the degree of infertility of some female soldiers. *Termopsis* is considered a very primitive genus on account of its many ancestral characters and its close resemblance to the even more primitive genus *Archotermopsis*. The castes of termites are regarded as segregants, arising by mendelian inheritance from a heterozygous parent form.

WYMAN, Boston.

THE CUTANEOUS WHITE LINE (OF SERGENT), SO-CALLED SUPRARENAL. SÉZARY, *Ann. de méd.* **11**:403 (May) 1922.

In 1904, Sergent described a sign which he considered characteristic of suprarenal insufficiency, namely, a white line produced by light stroking of the anterior abdominal wall. The present article continues the discussion of the validity of this sign with the following conclusions:

There is only one so-called white cutaneous line, and it is the physiologic white line described by Marey in 1858. It is found among healthy as well as among sick subjects. Whether associated with asthenia or arterial hypotension, this white line has no connection with suprarenal insufficiency. Its frequent occurrence in vigorous persons and its existence in patients in whom necropsy later reveals normal suprarenal glands, do not permit any doubt on this point.

The white line presents various modalities. It is, according to the case, sometimes intense and persistent, sometimes slight, of short duration or inconstant, sometimes absent. This diversity of a vascular reaction without doubt depends on the tonus of smooth muscle fibers and on the sympathetic system.

DAVIS, New York.

THE EFFECT OF THE EXTRACT OF THE POSTERIOR LOBE OF THE PITUITARY ON BASAL METABOLISM IN NORMAL INDIVIDUALS AND IN THOSE WITH ENDOCRINE DISTURBANCES. C. A. McKINLAY, *Arch. Int. Med.* **28**:703 (Dec.) 1921.

McKinlay determined the basal metabolic rate in twelve normal persons before and after the subcutaneous injection of the extract of the posterior lobe and pars intermedia of the pituitary. Eleven of the twelve subjects showed an increased basal metabolism, the percentile increase averaging 5. In four cases of hypothyroidism there was a diminution of basal metabolism following the injection of pituitary extract. In four cases of subnormal basal metabolism in which the influence of the thyroid was not suggested, injection was followed by a positive response. In four normal persons to whom thyroxin was given intravenously followed in a week by pituitary extract, there was an increase in basal metabolism of 8 per cent. In four other normal persons who did not receive thyroxin the increase amounted to 6 per cent. The author suggests that pituitary extract is effective in accelerating heat production only in the presence of a normally functioning thyroid gland, and that there is probably a synergic action between pituitary extract and thyroxin.

VONDERAHE, Philadelphia.

POINTS OF VIEW CONCERNING FOREBRAIN MORPHOLOGY IN LOWER VERTEBRATES. NILS HOLMGREN, *Jour. Comp. Neurol.* **34**: 391, 1922.

This paper brings out important new facts relating to the most primitive differentiation of primordia of the cerebral cortex in lower vertebrates. The mammalian cortex has three main divisions: hippocampal cortex, general cortex and pyriform cortex. Hitherto there has been no clear recognition of all of these divisions in any animals lower than reptiles; but Holmgren finds in the chief groups of fishes regions of the forebrain which he regards as points of departure for the differentiation of all of these cortical regions. This comes out most clearly in late embryonic stages of elasmobranchs. These observations open up an entirely new field of inquiry in comparative neurology, and may require considerable recasting of current ideas regarding the functional and morphogenetic factors involved in the separation of the cortical from the more ancient subcortical cerebral centers. And these are basic questions in the consideration of the still unsolved problems of human cortical function.

The paper contains also a review of the forebrains of lower vertebrates from the new point of view with an extended discussion of probable homologies of their parts. In conclusion, the problems of forebrain phylogeny are discussed in the light of comparative anatomy and embryology and of palaeontology.

C. J. HERRICK, Chicago.

DIAGNOSTIC VALUE OF SPINAL FLUID WASSERMANN REACTION. CESTAN and RISER, *Ann. de méd.* **11**:365 (May) 1922.

Vascular cerebral syphilis with meningeal involvement is often difficult to differentiate from paresis, even after a study of the spinal fluid. The authors report the results of an inquiry into the question of whether the spinal fluid Wassermann test performed quantitatively can aid in the differential diagnosis of certain forms of neurosyphilis. The report is based on the study of eighty-two cases of paresis, cerebral arteritis, syphilitic meningitis, tabes and syphilitic meningomyelitis.

In each case several examinations of the spinal fluid were made, extending frequently over a period of several months. The effect of treatment on the fluid was also studied. In each examination the Wassermann reaction was made with different quantities of fluid. With the other reagents constant, the smallest quantity of spinal fluid yielding a strongly positive reaction was called the positive index.

The authors conclude that it is not possible to make a differential diagnosis of the various forms of neurosyphilis on the intensity of the Wassermann reaction in the spinal fluid.

HYSLOP, New York.

CHANGES OF GOLGI'S APPARATUS IN NERVE CELLS OF THE SPINAL CORD FOLLOWING EXPOSURE TO COLD. C. DA FANO, *J. Nerv. & Ment. Dis.* **53**:353 (May) 1921.

The author studied the spinal cords of white rats exposed from two to twenty-two days to cold outdoor temperature. The supply of food was unlimited, and the animals continued to maintain their normal body temperature except when the cold was made more intense or prolonged. Changes were noted in Golgi's internal apparatus in the nerve cells of the dorsal horns, intermedio-

lateral and middle cell column and in the gray matter surrounding the central canal. No apparent changes were noted in the motor cells of the anterior horn. The characteristic of the change was found to be a tendency of the apparatus to assume a more robust aspect and to occupy a "tight perinuclear situation." In some of the cells canaliculi and spaces appeared in the cytoplasm similar to the trophospongium described by Holmgren. In the cases in which the exposure to cold had been such as to lower considerably the body temperature, disintegration of the apparatus was noted.

VONDERAHE, Philadelphia.

DECEREBRATE RIGIDITY IN ANIMALS AND ITS RECOGNITION IN MAN. F. M. R. WALSHE, *Proc. Roy. Soc. Med.* **15**:41, 1922.

After a brief review of the anatomy and physiology of decerebrate rigidity as defined by the work of Sherrington and Magnus, distinctions are drawn between this state of exaggerated tonus of extensor muscles which oppose gravity in the standing position, incapable of modification by other reflex mechanisms, and the conditions which result from removal of the brain just behind or in front of the thalami. In the latter condition tonus is not altered, and the animal can perform reflexly many complex motor adjustments.

Walshe then points out that the posture of extensor rigidity is not sufficient to establish a diagnosis of decerebrate rigidity in man, as it may be brought about by various lesions. He recognizes the fact that true decerebrate rigidity necessarily arises with the greatest frequency in midbrain lesions but may also be caused by damage to the reflex paths concerned and their control as the result of lesions higher up or in the spinal cord. Most of the cases described in the literature do not conform with the criteria laid down by Sherrington and should be excluded from this category.

SINGER, Chicago.

BLOOD ANALYSES IN CASES OF CATATONIC DEMENTIA PRAECOX. S. UYEMATSU and T. SODA, *J. Nerv. & Ment. Dis.* **53**:367 (May) 1921.

The authors select as the basis of their study catatonic cases of dementia praecox because they form a characteristic group and are readily differentiated. Systemic complications and metabolic disturbances were excluded. Control experiments were made on normal blood, and the nonprotein nitrogenous constituents and sugar were determined in thirty-two cases. None of the constituents showed any constant increase or decrease, but the average limits of the amounts of each constituent varied more widely than in the normal; this is interpreted as suggesting an unstable metabolic activity. In 47 per cent. of the cases blood sugar was increased beyond that of the highest normal reading, while in 75 per cent. of the cases a decrease in uric acid was noted; these patients, however, were inactive.

VONDERAHE, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 19, 1922

F. H. PACKARD, M.D., *President, in the Chair*

DEMONSTRATION OF A CASE OF PROGRESSIVE MUSCULAR ATROPHY. DR. J. W. COURTNEY.

The patient whom I am about to show is a striking instance of the scapulohumeral type of progressive muscular atrophy. The disorder made its debut on the right side, fifteen years ago. The patient was first seen by me at the Carney Hospital twelve years ago. He was then given a certain number of subcutaneous injections of strychnin. Since that time his treatment has been nil. The wasting of the right shoulder-girdle and arm is now practically complete. The forearm and hand are untouched.

My sole purpose in bringing this man before you is to afford an ocular demonstration of the slowness of the spinal cord decay, even under the most favorable conditions. The patient is a native of the West Indies, 52 years of age, married and the father of five children. From the beginning of his disorder, practically up to the present time, he has worked uninterruptedly as a cook, either in a private capacity or for railroad construction gangs. Only this autumn the left scapulohumeral region began to show evidence of involvement, and the patient now, for the first time, finds his ability to maintain his family in jeopardy. Already the fibrillary twitching of the left shoulder girdle groups is extreme. The left forearm and hand are intact. There is no atrophy elsewhere.

Patients of this kind seldom continue attendance for long at any one clinic; hence, a definite knowledge of their ultimate history is rarely available.

A CASE OF TRAUMATIC ABSCESS OF THE ANTERIOR CEREBRAL FOSSA. DR. J. W. COURTNEY.

My second case is a striking demonstration of the value of the temperature as a guide to the nature of the pathologic sequelae of head injuries. A boy, aged 7 years, on August 10 of the present year was hit in the face by an automobile. Half an hour after receiving his injuries, he was seen by several members of the staff of the hospital at Clinton, Mass. He was conscious. His face was swollen, both orbital regions were ecchymotic, there was an abrasion and hematoma over the left frontal bone, a small laceration on the chin, a fresh defect in the hard palate, a flattening of the bridge of the nose and a tearing away from the jaw of the upper lip and the nose, thereby exposing certain unerupted teeth. There was considerable hemorrhage. The wounds were immediately explored and carefully dressed, but nothing further was done until Aug. 14 when, under light ether anesthetization, the bridge of the nose was elevated. At this time firm pressure was insufficient to correct the defect in the hard palate, so no sutures were taken. The boy stood the operation well and continued to improve from that time on, showing no paralyses, no disturbance in the fundus oculi and no alterations in the

reflexes. The temperature was the characteristic one of *contusio cerebri*, that is, it alternated from normal to about 101.5 F. during the nineteen days of the patient's stay in the hospital. During this time a roentgen-ray examination revealed a linear fracture extending from the region of the roof of the mouth upward in the median line to the ethmoid region, where it deviated obliquely to the left, involving 3 inches (7.62 cm.) of the left frontal bone.

After leaving the hospital, the boy had a temperature of 99.5 to 100 F. for two weeks. From that time on his temperature was normal until ten days prior to his second admission to the hospital on Oct. 7. He had continued under a nurse's care, but he was up and about and had no symptoms. Rather abruptly on October 4 or 5 he developed a temperature of 102 to 103 F., and on October 6 became drowsy and irritable and complained somewhat of headache. The white cell count was 16,000. Now, to retrace my steps a bit: There was noted on September 23 a mucopurulent, bloodstreaked, discharge from both nostrils; but examination of the nose and transillumination of the sinuses revealed nothing significant. Both fundi were normal at that time. On September 29, examination of the latter disclosed a slight engorgement of the blood vessels, and on October 6, the day before I saw the boy, there was a slight choking of the left disk, with moderate engorgement and tortuosity of the vessels of the right fundus.

I examined the patient at the hospital on October 7. He was perfectly conscious, but flushed and inclined to somnolency. The pupils were widely dilated but equal and sluggish to light and accommodation. The fundus was *in statu quo*. There was no disfiguration of the face, with the exception of a slight flattening above the left superciliary ridge. There was no paralysis of any degree. The reflexes were unaltered. The nose and ears disclosed nothing abnormal. The heart sounds were clear, but there was a distinct intermittence of the pulse, which had not been noted before.

In making a diagnosis I had clearly in mind the favorable channel afforded by the fracture of the nose for the entry of pathogenic organisms. I was also much impressed by the sudden sharp rise of temperature, after a temperature-free period of many days. On these grounds I assumed the presence of an abscess in the anterior chamber of the skull, but from much discouraging previous experience, I was not enthusiastic about operation. However, I advised interference, if within the next few hours the boy's somnolence deepened. He did, in fact, not only grow more stuporous, but developed a ptosis of the left upper eyelid; so the next day about noon, under light etherization, a transverse incision was made through the left eyebrow, and the sinus was opened. This contained a little mucopurulent material and showed a linear fracture in its floor. Its posterior wall was then cut away in an area about one-quarter inch (6.35 mm.) in diameter. The frontal lobe pressed forward into the opening and did not pulsate. The dura was incised and a probe carefully passed around under it. The probe was then passed along the floor of the skull for a short distance, whereupon there was a gush of thick, very foul-smelling pus to the amount of about 4 ounces (113.4 gm.). At this the frontal lobe was observed to pulsate. Irrigation was then done, a rubber dam drain inserted and the wound closed with catgut.

Following the operation, improvement in the patient's general condition was noted for a time, but within a few hours the temperature began to climb to the regions of 105 and 106 F., and clonic spasms appeared in the right side of the face and right arm. These phenomena continued until death

about 10:30 the following day. At the moment of death the temperature was 108 F. A culture from the abscess showed no growth after twenty-four hours.

I have observed a number of cases of cerebral abscess of varied etiology. Several of these were evacuated with skill and dispatch. I have yet to see one patient survive the operation.

In the case of progressive muscular atrophy shown by me this evening, as well as in all others presenting chronic decay of the central nervous system, there is much food for reflection. They are tragic reminders of the utter futility of the necessarily empiric therapy we apply to them. How may we expect to be relieved of this embarrassing situation? In my own professional life I have followed the growth of the literature concerning their minute pathology to its present appalling proportions. It is an edifying testimonial to unrelenting and meticulously painstaking industry and to inexhaustible patience. But, unfortunately, as it now stands, it is no more vital to the achievement of a scientific therapy than the tissues whence it was derived. To my way of thinking, the data contained in this enormous literature demand to be assayed, so to speak, by a genuinely scholarly mind of the Huxley order. By this I mean a mind which is utterly free from emotional bias; which possesses so truly the gift of sagacious discernment that it never by any chance confounds differences in kind with differences in degree; which is capable of observing and generalizing phenomena and of expressing with punctilious accuracy their order and sequences; and which, withal, is stimulated, and, at the same time, nicely controlled by a sane and vigorous imagination. An assay made by such a mind is bound to yield a universally intelligible conception, not only of the nature of the morbid agents which effect the decay of the nervous elements involved in the diseases in question, but also of their *modus operandi* at every stage of the process. When it is accomplished, the clinician may hope to come into possession of a scientific program of prophylaxis and treatment.

THE PHYSIOLOGY OF AUTOSUGGESTION. DR. DONALD GREGG.

At this time when "autosuggestion" is receiving wide publicity, the subject may well be given consideration by the physician as well as by the layman. Although the physician may attempt to laugh the matter out of court, the layman continues to say, "But there is something in it just the same."

Suggestion implies the passing of an idea from one to another. Without a "conscious" and an "unconscious" autosuggestion becomes impossible. Dr. Morton Prince, whose book on the "Subconscious," popularized the term, has said that there is no such thing as "the subconscious" as distinguished from "the conscious." Dr. Coué in his booklet supplies no proof. He refers to somnambulism, delirious conduct, unconscious memory and control of the autonomic system, all of which phenomena can be explained without considering the unconscious. Most of Coué's thesis regarding self-mastery centers about the control of physical functions through conscious suggestions that are thought to be absorbed by the unconscious, which, in turn, control the bodily functions and produce health or disease. Such a thesis, if tenable, should be applicable in sickness and in health, in infancy and old age, and in the past as well as in the present development of man. What becomes of the unconscious in mental disease? At what age does it begin to develop, and when in man's development did he acquire an unconscious self? If we simply regard the body as a mechanism which automatically runs along healthily in the great majority of cases unless interfered with by noxious agents from without, or unless its activities are handicapped by malformations or maldevelopments, or its functions inhibited or disturbed by the emotions, have we not an unassailable thesis? Without recourse to the confused and disputed terms of conscious and unconscious, we have already in

the intellect and the emotions a duality of functions that explains the situation. We recognize that the emotions modify our vegetative activities, often contrary to our intellect and will, which have little to do with these primitive functions. Here, then, is the physiologic explanation of "autosuggestion" so called. And he who in functional disease heals the sick or helps himself to health, does so, not through the unconscious, but through the action of the emotions. By eliminating the inhibiting or disturbing actions of the emotions in their multifarious forms, functional health is often regained, and he who would heal himself or others needs to know the physiology of the emotions, and not the pseudopsychology of the unconscious.

DISCUSSION

DR. GEORGE A. WATERMAN: The matter is more complex than the division of these reactions on the basis of emotion and intellectual processes only would imply. It is difficult to explain many of the phenomena which are of everyday occurrence, and one is surprised at the number of phenomena that happen in nonpathologic cases. In the every-day life of many of us we find phenomena which, after lying latent in the subconscious, may rise to the conscious, as in the dream states, for instance. So frequently one will wake up and say he has not dreamed at all, when some sudden sound or seeing some object will cause the dream to flash into the mind. These things in the subconscious have an effect on the activities of the person, though he is entirely unaware of their existence. This was illustrated by a young woman who said she thought she had not dreamed, but whose dream was instantly recalled to her mind by seeing a black evening gown hanging in the closet. She had worn the gown to a ball at which she had seen the man of whom she dreamed.

Hypnotic suggestion given in the sleep state produces mental activity without the patient's knowing the cause. I think there can be little doubt that there are activities beneath the threshold of thinking that may produce activities in the intellect. The terminology should be more definitely settled.

RECENT DEVELOPMENTS IN ELECTRO-DIAGNOSIS. DR. PERCIVAL BAILEY.

This article will be published in full in a future issue of the ARCHIVES.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 27, 1922

C. H. FRAZIER, M.D., *President, in the Chair*

A CASE OF MYASTHENIA GRAVIS OF SLOW DEVELOPMENT. DRS. W. G. SPILLER and M. G. CHADMAN.

The interesting features of this case are the long duration of the symptoms, dating back fourteen years with gradual progression, the comparatively slight implication of the ocular muscles, which usually are among those most affected in myasthenia gravis, and the results of the creatin-creatinin study by Dr. Jonas.

A woman, aged 41, a Hungarian, in 1908 noticed that if she danced for a few hours her legs would become tired and tremble, but after a short rest they would feel well again. By avoiding undue exercise she had no further

symptoms until six or seven years later, when her voice "broke" after a rather lengthy reading; for a few minutes she was unable to speak, but after a short rest her speech returned. The cause of the dysarthria seemed to be a weakness of the lingual and palatal muscles, so that she was unable to form words. About a year later she first experienced difficulty in swallowing, the food lodging in her throat. She could not take solid food and had to live on liquids for a week. For the next four years there was no progression of the symptoms, the only trouble being inability to speak longer than a few minutes at a time, and a feeling of fatigue in the muscles of mastication toward the end of a meal. Within the last two years she has had more difficulty in swallowing. As a meal progresses her jaws become tired and she is unable to swallow solid food, which causes her to cough, after which the bolus is regurgitated. Liquids never cause difficulty in swallowing. About this time she noticed that her limbs would tire quickly after moderate exertion. After riding in an automobile a short distance she would be unable to get up and walk. If she would write for a short time she would have to stop and rest because of muscular fatigue. She then noticed that her eyes would tire on reading and she would see double. To avoid this she read with one eye covered.

At present she has trouble keeping her eyes open, and the difficulty becomes greater as the day advances. She has to exert a constant effort to keep them open and often sits with the head tilted back and eyelids partly closed. She cannot write a postcard without resting, and has to stop and rest several times during each meal. Walking the length of the ward tires her slightly. Her back feels weak and tired. There is no pain in the affected muscles, only a tired feeling after exertion. The hands, forearms, feet and legs feel numb at times. If her hands get cold they become so numb and stiff that she cannot use them. There are no other subjective sensory disturbances.

Physical Examination.—There was no weakness of the ocular muscles except a slight ptosis of the lids. There was slight weakness of the facial muscles, the forehead being wrinkled poorly, the eyelids closing weakly, and the facial lines being poorly marked on smiling and whistling. The muscle power was fair but repeated effort soon caused feeble response, and the movements then became incoordinate. The reflexes (biceps, triceps, patellar and Achilles) were normal and equal on both sides.

After rest speech was clear, but the patient made an obvious effort to control the tongue, lips and palate. After a short conversation the efforts became pronounced, and syllables were not pronounced distinctly. The tongue often appeared to stick, and the patient had to repeat the attempt to pronounce a syllable.

On swallowing, the bolus of food often caught in the throat, and the patient had to make several attempts to swallow it and often failed, beginning to cough and then regurgitating the bolus.

Eye Examination.—The right palpebral fissure measured 7.5 mm., the left 7 mm., becoming slightly narrower as fixation was prolonged. The ocular rotations were full, without diplopia. The media were clear; the disks were slightly oval and healthy large shelving temporal cups. No change in the choroid or retina was noted.

Electrical Examination.—The muscles tested were the orbicularis palpebrarum, orbicularis oris and flexor communis digitorum, the method used being a rapidly interrupted faradic current (tetanizing in effect) which entered the muscles at their respective motor points. A sharp tonic contraction ensued which at the end of about fifteen to thirty seconds began to relax in spite of

the continued excitation. (Comparison was made with a normal muscle in which the current was allowed to flow for five minutes with no sign of relaxation of the tonic contraction.) Repeated galvanic stimulation resulted in sharp responses of a progressively diminishing amplitude of contraction until the muscle would not respond without a rest period. A moderately rapid faradic stimulation (250 per minute) demonstrated failures of contractions.

DISCUSSION

DR. LEON JONAS: During the last decade, there have appeared numerous papers pertaining to biochemical studies in myasthenia gravis. The abnormalities of metabolism centered on three substances: calcium, creatin and creatinin. Certain workers, notably Pemberton and Spiller, found a calcium loss; others found no change in the calcium balance; and a third group reported a calcium retention. A case in which I investigated the metabolism for Dr. Spiller in 1918 showed a slight calcium retention. In view of the conflicting calcium results, it would seem that alterations of calcium metabolism have no particular significance in myasthenia gravis.

Before discussing creatin and creatinin change, I shall briefly explain the normal physiology of these substances. Creatin and its anhydrid creatinin are related to muscle metabolism, and it has been suggested that the urinary output of these substances is in proportion to the muscle bulk. One must be guarded in one's statements on creatin and creatinin metabolism because of the conflicting views on certain phases of this subject.

The creatin coefficient is the number of milligrams of creatinin nitrogen per kilo of body weight. This is approximately 8 in men, but is lower in women. Creatinin is a normal constituent in the urine and is very constant when the subject is on a creatinin-free and a creatin-free diet. Creatin is found in the urine until the eleventh year. It may appear in the urine after this age under the following conditions: first, a low carbohydrate tolerance or insufficient intake of carbohydrate, as in diabetes starvation; second, overstimulation of metabolism as in fever, hyperthyroidism and high protein feeding; third, muscular wastage as in the dystrophies, and when the ability to store creatin is not equal to the supply, as in children. Creatin appears during pregnancy, puerperium and at times during menstruation.

In myasthenia gravis all workers report a low creatinin output. Creatin has been reported by some workers in the urine of patients suffering from this disease. Williams and Dyke found a creatinuria in four cases recently reported in the *Quarterly Journal of Medicine*. Our patient showed no creatinuria. The cases of Williams and Dyke showed a lowered carbohydrate tolerance when tested by giving 100 gm. of glucose and studying the blood sugar curve.

In a normal person, after taking 100 gm. of glucose the blood sugar should not be higher than 150 mg. per 100 c.c. of blood, and sugar should not appear in the urine. The threshold for glucose is 170 mg. per 100 c.c. of blood. In the cases of Williams and Dyke the height of the blood sugar curves were above 170 mg. In the case reported the blood sugar was not more than 123 mg. after giving 100 gm. of glucose, and no glucose was found in the urine.

In view of the association of the impairment of the carbohydrate tolerance in the cases of Williams and Dyke with the presence of creatin, the absence of creatinuria in our case may have some relationship with the normal or rather increased tolerance for glucose.

DR. FRANCIS X. DERCUM: Before the patient departs I would like to call attention to the nasal intonation of the patient's speech due to the involve-

ment of the soft palate and also the marked accentuation of both nasolabial folds which gives an expression suggesting the risus sardonicus. In my experience this deepening of the facial folds gives an almost characteristic facial expression to patients with myasthenia gravis.

THE ASSOCIATION OF HERPES ZOSTER AND TABES DORSALIS.

DR. W. B. CADWALADER.

The occurrence of herpes zoster in tabetic patients or in patients suffering from cerebrospinal syphilis does not seem to be common. Head and Campbell (*Brain*, No. 3, 1900, p. 366) found that the alterations that take place in the posterior spinal ganglions and in the gasserian ganglion of patients with herpes zoster of syphilitic origin resemble those found in other conditions. These changes are: (1) inflammation with exudation of small deeply staining cells, (2) extravasation of the blood, (3) destruction of ganglion cells and fibers, and (4) inflammation of the sheath of the ganglions. The authors state that in tabes dorsalis outbursts of zoster form a classical symptom, but that this phenomenon is rarer than textbooks would lead one to believe. They also state that paresis predisposes to zoster, and they were inclined to attribute it to the arterial changes that take place in the ganglions and not necessarily to the primary syphilitic process.

The involvement of the sympathetic nerves to the eye causing sympathetic ophthalmoplegia that occurred in my second case can be attributed to the syphilitic process affecting the sympathetic nerves that pass close to the ophthalmic division of the trifacial nerves in its course to the gasserian ganglion. Dr. Dorrance, in a paper on alcoholic injections of the gasserian ganglion, presented before this Society a few years ago, called attention to the fact that this produced flushing of the face and lachrymation—symptoms of sympathetic involvement. The severe pain in the eyeball with lachrymation and photophobia that also occurred in my second case greatly resembled the so-called "optic crises of tabes." Dr. Spiller made a special report of such a case before this Society a number of years ago.

CASE 1.—A woman, 60 years old, during the summer of 1921 developed considerable pain in the right upper limb, quickly followed by a typical herpetic eruption on the outer side of the upper arm and of the forearm. Soon after this she began to have lancinating pains in the lower limbs followed by diplopia and by ataxia in walking, and also girdle sensations.

Physical examination revealed the usual signs of tabes dorsalis, and Dr. deSchweinitz reported that both fundi were normal. The Wassermann reaction was positive.

CASE 2.—A man, 65 years old, was healthy until July, 1922, when he suddenly developed severe burning pain in the left eyeball with much lachrymation. The pain later extended through the left side of the nose and over the left forehead. After a few days had elapsed, a typical herpetic eruption appeared throughout the distribution of the ophthalmic division of the fifth nerve. Except for these symptoms, he felt fairly well.

Physical Examination.—The right pupil was small and reacted slowly to light and in accommodation. Ocular movements were normal. The pupil of the left eye was contracted, and the left palpebral fissure was narrower than the right. The left cornea was anesthetic; there was hyperalgesia throughout the distribution of the ophthalmic division of the fifth nerve on the left side. In this distribution scars of the herpetic eruption were still present. Pain, though

not nearly so severe as formerly, had not disappeared. Dr. deSchweinitz reported that the patient had left enophthalmos and slight myosis unaffected by cocaine. There were no fundus lesions, and the fields were normal. The gait and station were normal; tendon reflexes of the lower extremities were greatly diminished but not lost. The Wassermann reaction of the blood serum was negative. Sensation over the distal portions of the lower limbs for pin prick was slightly delayed; over the thorax it was normal.

DISCUSSION

DR. SPILLER: In 1893 or 1894 I saw a woman who had cervical Pott's disease. She had herpes zoster in the distribution of the second thoracic root only on one side. At the necropsy the first thoracic root was removed by mistake for the second, and it was entirely normal. The second thoracic root when removed was found to be covered with miliary tubercles. This is the first time to my knowledge that a distinct relation of herpes zoster to disease of the spinal ganglion has been established.

DR. CHARLES H. FRAZIER: Frequently after operations on the gasserian ganglion herpetic eruptions occur, possibly one in every three cases, but so far as I remember I do not recall having seen any in the first division. The great majority were in the second division, the others in the third; they were almost invariably on the lips.

AN ACUTE ALCOHOLIC PSYCHOSIS ASSOCIATED WITH BRAIN TUMOR. DR. C. W. BURR.

A white man, 51 years old, went to the Philadelphia Hospital on Sept. 5, 1922, complaining of headache. There was a superficial wound 2 inches (5.08 cm.) in length above the right temple. He said that he had been hit with a hammer, but he was so confused that he could not give any details of the injury. He said that he had been drinking hard for two weeks. When put to bed he became more confused, picked at the bed clothes, rocked back and forth and snapped his fingers. He seemed to have particular difficulty in recalling words. He was left-handed. A few days later a relative was found who said that the patient had been a wanderer for the last few years, previous to which he had been a professional ball player. His habits previous to leaving home had been good, and the relative said that he had always acted normally, although she had not seen him for several years.

Physical Examination.—The restlessness present on coming to the hospital decreased, but the man continued to be dazed. When asked a question he would stare at the questioner and repeat the words several times. Sometimes he would finally give an answer, usually relevant; sometimes he would not reply. There was no paralysis of the arms, legs, face or tongue. He had no difficulty in swallowing and no disturbance of articulation, but quite frequently he was unable to recall a word. His abdominal and thoracic organs were normal. There was no disturbance of any of the reflexes, and the gait was normal. He was careless about his bladder and rectum and unconcerned about his habits. At times he was irritable, at times quiet. He adopted several muscle habits, for example, he would stir an empty pan with a spoon for many minutes at a time and would rub a dish back and forth on the table. On October 12 he had a general epileptiform convulsion, after which he slowly sank into coma and died eight days later, having been in a coma throughout that entire period. Both the blood and spinal fluid Was-

sermann reactions were negative. Roentgen-ray examination of the head showed no fracture. Dr. Langdon reported that the pupils responded well to light and accommodation, that the ocular rotations were full and equal, and that there was no nystagmus. The ocular arteries were small, but there were no signs suggesting sclerosis. The disks showed some loss of capillarity without other fundus changes.

During the first two weeks his temperature was a little subnormal, during the following four weeks it was normal, and during the balance of the time it ranged from 99 to 102.5 F.

At necropsy a glioma of the right frontal lobe was found and in addition, chronic myocarditis, chronic endocarditis, splenitis, interstitial nephritis and chronic perihepatitis. The tumor mass had no wall, infiltrated the nerve matter, and there was a hemorrhage within its degenerated center.

I doubt if the tumor caused the symptoms. The mental picture was that of chronic cerebral alcoholic poisoning. Headache was the only symptom of tumor of which he complained, and there were no physical signs of it. The one convulsion may have occurred at the time of the hemorrhage.

THE AVELIS SYNDROME. DR. ALFRED GORDON.

A man, 38 years of age, became ill about three months ago, while getting off a car, when he noticed that he inclined toward the left when walking, although this symptom lasted only a few minutes. Several days later he suddenly developed severe headache in the right temporoparietal region, which persisted several hours unaccompanied by vomiting or dizziness. The next morning he felt very weak and could not go to work. The headache reappeared on the same side and was accompanied by vertigo, both lasting from five to ten minutes. This was followed immediately by difficulty in swallowing and awkwardness in the left upper limb. At the end of five weeks he presented the following condition: The gait and station with eyes open or closed were normal. The movements of the extremities were correct, and the grip of both hands was normal. The head was slightly tilted to the right, and the face seemed slightly deviated to the left. The right orbital fissure was wider than the left. The pupils were equal and reacted to light, but the right responded more sluggishly than the left. The ocular muscles and eyegrounds were normal.

There was no ataxia, adiadokokinesia or tremor in the upper extremities. The knee jerks were markedly increased; there was no ankle clonus, and the plantar reflexes were of the flexor type.

Sensation was altered on the left side of the face (in the distribution of the second and third branches of the fifth nerve), neck, thorax as low as the mammillary line, and on the left shoulder, arm and hand; in this area the sense of touch was normal, but pain and temperature perceptions were considerably diminished, cold being taken for warm, and hot was in some places not recognized at all. The deep sensibility on the left side was not involved. Another conspicuous symptom was the inability to swallow liquid or solid food, so that the patient had to be fed through a nasal tube. The soft palate and uvula, the epiglottis and entire larynx appeared drawn to the left. The vocal chords functioned normally; sensation over the right side of the soft palate and uvula was greatly diminished. The sense of taste, the tongue and hearing were normal. A vestibular test showed loss of function of the vestibulo-ocular as well as of the vestibulo-cerebello-cerebral pathway from the vertical semicircular canals of both sides. The horizontal canal

pathway appeared to be unaffected. The Wassermann reaction of the blood and spinal fluid were normal. His mentality was good. Urinalysis was negative. The heart was somewhat enlarged, and a mild presystolic murmur was present at the base.

Repeated examinations of the patient invariably gave the same results. Only a few days ago some modifications were observed for the first time. The syringomyelic sensory dissociation described in the foregoing became less marked on the left side of the face. The area of the second and third branches of the left trigeminus became less affected; instead of complete loss of pain and temperature perception, there was merely a slight diminution of these two sensibilities, and the real loss of sensations commenced at the lower border of the mandible. Moreover, the deviation of the face to the left was also less marked than before. The patient had recently observed that small quantities of fluid passed down the esophagus without much difficulty.

The most conspicuous symptoms in this case were the difficulty in swallowing and the sensory dissociation on the face and left arm and thorax. The lesion evidently lay in the medulla, involving the nuclei of the ninth, tenth and seventh nerves, also Gowers' tract. The lesion was evidently vascular and consisted of an occlusion of the posterior inferior cerebellar artery. The case is interesting from the standpoint of the limited number of symptoms, which is contrary to what is found in the majority of cases of this character.

DISCUSSION

DR. W. B. CADWALADER: The distribution of sensory disturbances in Dr. Gordon's patient appear to be most unusual, for the anesthesia was on the face and limbs of the side opposite to the lesion. Usually in cases of thrombosis of the posterior inferior cerebellar artery there is disturbance of sensation of the face on the same side as the lesion in the medulla oblongata, with disturbance of sensation of the limbs on the opposite side.

DR. SPILLER: The explanation of the symptoms in Dr. Gordon's case, it seems to me, is to be found in a lesion of the right side of the medulla oblongata, the side on which the paralysis of the soft palate occurred. The disturbance of pain and temperature sensations on the left side of the face is to be explained by implication of the central fibers passing from the nucleus of the left spinal root of the fifth nerve and after decussation ascending on the right side of the medulla oblongata. The limited implication of pain and temperature sensations on the left side of the trunk extending only a short distance downward indicates that the fibers in the spinothalamic tract from only the part in connection with this distribution were implicated on the right side.

CLINICAL AND PATHOLOGIC DIFFERENTIATION OF THE SENILE PSYCHOSES AND THE ARTERIOSCLEROTIC PSYCHOSES. DR. A. R. VONDERAHE.

The psychoses peculiar to old age, according to the current classification, are divided into two groups: the senile psychoses and the arteriosclerotic psychoses. Some clinicians do not follow this grouping but consider the term "senile dementia" adequate for both types; others include both under such a term as "arteriosclerosis of the nerve centers."

A separation into senile psychoses and arteriosclerotic psychoses, however, can be made on the basis of clinical symptomatology. An analysis of eight

consecutive cases from the Pennsylvania Hospital was made. Seven of these cases were classed as senile psychosis; one as arteriosclerotic psychosis. The differentiation rests on excluding the symptoms characteristic of cerebral arteriosclerosis, such as headaches, dizziness, feelings of pressure, syncopal attacks, and particularly the finding of some focal sign referable to an arteriosclerotic process. In the seven cases diagnosed as senile psychosis the mental symptoms were found to be much the same in all: impairment of memory, disorientation, confusion. In addition, in one case there were visual and tactile hallucinations; in another a state of apprehension with delusions of infidelity, and in another a tendency to maniacal attacks. Physical examination in each instance showed evidence of cardiac impairment, either hypertrophy, irregularity or a murmur or a combination of these. In all the cases diagnosed as senile psychosis there was sclerosis of both radial and temporal arteries with tortuosity; in two instances there were unequal pupils; in one instance the pupils were irregular and did not react to light; in one instance, in which the eyegrounds were examined, arteriosclerosis and tortuosity of the retinal vessels were found. The blood pressure was not constantly increased. In every instance but one the urine contained albumin and hyaline or granular casts. In the cases which were diagnosed as senile psychosis, accordingly, renal and myocardial changes were found to be prominent in the symptomatology.

The views of Kraepelin, Bing, Alzheimer, Nötzi, Meyer and Appledorn regarding the gross and microscopic pathology of the condition, vary. Southard had hoped to establish a case of pure senile atrophy but was not able to exclude arteriosclerosis entirely in any one instance.

The available pathologic material and clinical records at the Philadelphia General Hospital were studied. In a total of 435 brains, 189 showed arteriosclerotic changes. Of the total which showed arteriosclerosis, only twenty-five, or 13.2 per cent., were diagnosed clinically as senile dementia, thus illustrating the well-known observation that marked arteriosclerosis may occur in the cerebral vessels without psychosis.

On the basis of the differential points noted in the foregoing, the cases were grouped into senile psychoses and psychoses with cerebral arteriosclerosis. The gross and microscopic anatomy was studied. In all cases but one evidence of cerebral arteriosclerosis was evident grossly and microscopically. In the latter case, however, microscopic examination of the frontal, occipital, parietal and temporal cortex showed thickening of a patchy type in the smaller vessels, while the larger vessels apparently escaped; there was also thickening of the pia, some glial proliferation and evidence of retrograde processes in many of the cortical cells. The systemic pathology of this case is of interest. There was ulcerative arteriosclerosis of the aorta, sclerosis and calcification of the coronary arteries with myocardial degeneration. The kidney grossly showed evidence of chronic passive congestion and microscopically, arteriosclerosis and toxic changes. Clinically, the psychosis developed about a year before death and was characterized chiefly by incoherent speech and depression. On physical examination the heart was found to be enlarged to the left; there was a harsh systolic murmur at the apex, transmitted to the axilla and an occasional extrasystole.

Seven of the cases could be classed as senile psychoses. One of these cases has just been described. In the remaining six, cortical atrophy, pial thickening and gross arteriosclerosis of the basal vessels were found. Microscopically these brains showed cellular atrophy, diminution of the cells of

the outer layers of the cortex, increased glial proliferation, evidence of retrograde processes and lipid accumulation in one instance. Varying degrees of sclerosis of the smaller arteries were noted.

Twenty-five cases diagnosed as "senile dementia" from the Norristown necropsy reports were analyzed. In two instances in which the brain was described but no mention made of the arteries the visceral changes were noted in detail; in one there was found chronic aortic and mitral stenosis, coronary arteriosclerosis, chronic interstitial nephritis and cirrhosis of the liver; in the other, chronic interstitial myocarditis, mitral and aortic valvulitis, chronic atrophic gastritis, chronic cholecystitis, cirrhosis of the liver and chronic interstitial nephritis. From the same source it is found that eighty-four of ninety patients with senile dementia have nephritis in some form or other, sixty-two having chronic interstitial nephritis, seventeen chronic parenchymatous or chronic diffuse nephritis, and five suppurative nephritis.

In conclusion, it may be noted that the term senile dementia is sometimes used to include the senile psychoses and arteriosclerotic psychoses, but that a clinical separation into two such forms can be made and is useful. Pathologically, there is difficulty in separating the two. In cases diagnosed as senile psychosis, the consistent presence both clinically and pathologically of recognizable heart or kidney lesions is emphasized. Cases of senile atrophy with moderate arterial change, or at least with arterial change insufficient to satisfy the observer of a casual relationship to the atrophy, have been described. In such cases, the rôle of arteriosclerosis and other pathologic conditions in the viscera is suggested as forming a possible basis for a toxic neuronc degeneration of the cortex.

DISCUSSION

DR. CHARLES K. MILLS: As has been pointed out, in senile dementia, as recognized both clinically and pathologically, just as in cases which are definitely to be designated as cerebral arteriosclerosis, vascular disease is present. That fact was notable in some of the observations quoted by Dr. Meyer and others.

Two points seem to help most in the differential diagnosis of these affections. They depend on the question of the degree of the vascular disease and on the question of the locality of the lesion which is present. Many of the symptoms of senile dementia, as seen in both hospital and private practice, are due, not to destructive focal disease, but are dependent on the lack of a proper blood supply passing through vessels that are not sufficient in caliber normally to nourish the brain. Consequently, there is a general depression in cerebral function and a change in the mental phenomena presented by these patients. We get not only impairment of memory, both in arteriosclerosis, so-called, and in senile dementia, and perhaps a certain degree of disorientation, but in true senile dementia recognized as this psychosis, we have mental symptoms of peculiar type, some of which have been mentioned. We have hallucinations of particular type, and we have also what seems to me to be of particular importance for true senile dementia, a peculiar form of persecutory delusions—a sort of reversal of ordinary familial relations. In old age, in cases which are properly called senile dementia, the person suffering from this affection will turn from the members of his family who have always cared for him and helped him in every way, perhaps to some member of the family who has not taken any interest in him, or has even neglected him.

I have seen many, and some sad, cases of cerebral arteriosclerosis. I have had an opportunity to study them in private practice over a series of years, cases which have developed before a really general senile condition had arisen. The one thing that has led me to a positive diagnosis of cerebral arteriosclerosis, having in the first place excluded certain focal lesions like brain tumor and focalized meningitis, is the fact that there are symptoms of mental dissociation which are evidently due to focal lesions, especially in the deeper regions of the brain. After loss of memory, there is disorientation, often then apraxia, and next aphasia of the dissociating type. This aphasia becomes more marked until verbal incoherence is present. In my own experience, headache has not been so prominent a symptom. Curiously, in some cases patients who have suffered from extremely severe recurring sick headache, after the development of clear indications of cerebral arteriosclerosis, have had a diminution in the severity and in the frequency of the headaches. Headaches are, however, sometimes present, but I would not give them the importance assigned by the reader of the paper.

Areas of multiple softening will dissociate eventually one part of the brain from the other until the entire brain is dissociated, whereas in the cases of senile dementia, there is a general parenchymatous or nutritive disorder of the brain which is due to an insufficient blood supply, but which is not so frequently accompanied by focal lesions in connection with vascular disease.

LEFT HEMIPLEGIA WITH APHASIA IN A LEFT-HANDED PERSON,
AND WITH AGRAPHIA IN THE OTHERWISE UNAFFECTED
HAND. DR. ALBERT C. BUCKLEY.

The following case report is of unusual interest and therefore worthy of record, because it deals with an aphasic and agraphic left-sided hemiplegic patient who was naturally left-handed. She had educated the right hand for writing, but had lost the ability to write with the right hand when the left side became paralyzed.

The motor aphasia was at first complete; later marked improvement in speech followed, but the ability to write, up to the present time, nearly six months after the stroke, has not returned. The patient was naturally left-handed, that is, she always took the initiative with the left hand to grasp objects, and she sewed with the left hand. She had learned to knit and to write, however, with the right hand, and always used the right hand for writing.

The early medical history presents nothing of importance from the standpoint of the present illness. She had suffered from severe attacks of "indigestion," and was treated for gastric ulcer in 1904 and in 1908, from which she apparently recovered.

The course of the patient's disorder is as follows: While occupied with household duties, the patient was seized with a feeling of fatigue, difficulty in holding objects with her left hand, loss of speech, followed by total loss of power in the left hand, and finally paralysis in the left leg, in the order named. Physical examination revealed a large framed, middle-aged woman, who presented the general appearance of a person in robust health, weighing about 200 pounds (90.72 kg.). The pupils were unequal, the right larger and flattened on the superior margin. There was prompt iris reaction in both eyes to light stimulation and with convergence; consensual response was preserved in both eyes. There was no hemianopsia. There were no marked changes in the fundi; the disks were described as being paler than is usual for a

person of her age; the arteries were distinctly thickened and their caliber reduced. There was no ocular palsy, no weakness of the ocular muscles or nystagmus.

There were no other cranial nerve signs than the apparent weakness of the tongue causing it to deviate slightly to the left of the midline. The left side of the face was slightly flatter at the cheek, and distinct weakness of the same side was shown when the mouth was opened wide. The tongue deviated slightly to the left of the midline, but was protruded without weakness or tremor. The throat was negative although swallowing was difficult.

There was a total, flaccid, left-sided hemiplegia and hemianesthesia. The patient was unable to move in bed, lying on the left side by preference on account of pain in the region of the left flank. Passive movements of the paralyzed arm caused pain in the shoulder region of that side. The biceps jerks were present on both sides, those of the paralyzed side being more active. Both patellar reflexes were active, the left more than the right. The plantar reflexes were present on both sides; stroking the right sole produced the normal plantar flexion; on the left side there was a definite Babinski reaction.

All forms of sensibility were lost on the paralyzed side, except occasional pain reactions to deep pricks. Faradic stimulation caused movements of the arm and leg muscles with the production of painful sensations in the paralyzed limbs.

There was no disturbance of superficial or deep sensibility on the right side; objects placed in the hand were named promptly when the patient could find the word, and when not able to find the word she was able to indicate by pantomime the use of the object. From this it is inferred that there was no stereognostic disturbance and no apraxia on the right side and no disturbance of sensibility. Thus far the patient presented the physical signs of a left-sided hemiplegia with anesthesia, the right side being functionally intact from the standpoint of sensibility and motility. There was at first complete incontinence of the bowels and bladder, due to mental dulness. The patient was at first practically wordless, with the exception of an occasional word which was distinctly enunciated, as most of her attempts at word formation were either unintelligible or sounded like "baby talk" whenever spontaneous speech was attempted. Words pronounced for her could, however, be repeated by the patient at the time. Attempts to combine words she had just pronounced invariably resulted in failure.

There was no word deafness, as was shown by her ability to pronounce some words given her and her understanding of most of what was said to her. She was interested in stories read to her and later was able to convey in part, by gesture and a few words, the meaning of what had been read.

She followed commands, as already indicated, and carried them out. Unusual words such as "Oshkosh," and "Kalamazoo" were pronounced with difficulty, after they had been repeated several times, carefully separating the sound of each syllable.

She was not object blind as shown by the fact that she recognized the use of common objects, such as a knife, key and pencil. She clearly indicated that she recognized individual letters and numbers at sight, and could name some of them as digits but failed in compounds. She later was able to count spontaneously from 1 to 16; then said "18, 9, 10, 11." She recognized such numerals as 20 and 30, but when shown 203, said "20 and 3." A few familiar words were recognized and pronounced, for example, "4th of July."

The remarkable disturbance in this patient was the loss of ability to write with the unaffected hand she normally used for writing. The agraphia was complete, including attempts to write her name. At times the agraphia appeared to be due to inability to use the correct word. For example, when told to write "plate" she said "dish" and attempted to write "dish," but failed. She recognized that her writing did not look right and was greatly annoyed by the fact. She did better at writing numerals from dictation, but had difficulty with these. In attempting to write 13, she said "one, one, one, three" and wrote 1113. She was unable to write the names of the days of the week after she had learned to repeat them. Attempts at spontaneous writing were hopeless failures. Writing from copy was also practically impossible, as only an occasional word could be legibly written.

During the first six weeks there was considerable improvement in speech, her vocabulary slowly increasing so that some sentences were completely formed. Words were frequently badly pronounced and often a word could not be found to complete an idea.

The difficulty in swallowing gradually disappeared, but the paralysis, except for slight power of adduction of the leg, was unchanged except that con-

The image shows a handwritten specimen of the patient's writing. The text is written in cursive and reads "It is a bright day." The handwriting is somewhat slanted and the letters are connected, with some variations in stroke thickness and spacing, characteristic of a patient with agraphia.

Specimen of patient's writing. "It is a bright day."

tractures appeared. There was little improvement in sensibility. Deep painful stimuli were felt here and there, but the patient was unable to locate the sensation thus produced.

The agraphia persists at the present time, six months after the onset of the disorder, there being no improvement in the attempts to write.

In the literature I have thus far not been able to find a similar case. Claude (1910) reported an instance of complete hemiplegia on the right with apraxia on the left. There was no apraxia in my patient. Pitres (1884) recorded a right-sided paralysis without aphasia in which the patient is reported as unable to write with the right hand, but wrote freely with the left hand. Meyer (1908) reported a left-handed man with left hemiplegia who was unable to write words, who could write letters but not the letters asked for.

One of my left-handed patients, a woman 70 years of age, paretic on the right and astereognostic, had lost her ability to write with the right hand, and later learned easily to write well with the left hand. Another elderly woman, right-handed, with a powerless right hand and marked dysarthria, but not true aphasia, is totally unable to write with the left hand. Another of my patients, now a right-sided hemiplegic, a right-handed person, is not agraphic in the left hand, although she writes with much difficulty.

It is an interesting question whether the patient under discussion, if she had been right-handed would have been agraphic on the right. It might also give valuable data if we were to determine whether our hemiplegic patients show any impairment in original writing ability of the contralateral hand. All of us are able to write legibly to a certain extent with the untrained hand. Is this impaired, and to what extent, after a paralysis on the opposite side?

In attempting to explain the mode of producing the agraphia in this case several possibilities were considered: 1. Bilateral lesions can be reasonably excluded on account of the absence of any physical signs in the right arm or hand. 2. A lesion in the visuopsychic cortical region on the same side as the principal lesion is excluded on account of the absence of mind-blindness. With the exception of failure to read written phrases correctly there was no distinct involvement, except early, when the motor aphasia was so marked that she could not pronounce any word. She read names of objects and designated her recognition of those objects at the same time.* 3. A subcortical lesion sufficiently large to include the paths from the motor, the sensory and the visuopsychic areas can also be excluded on similar grounds.

For years we have been following the subject of aphasia with utmost respect for diagrams of hypothetical construction, on which our explanations of speech disturbances in particular were based. More recently came the revisions of the subject by Marie and Bernheim. It seems that much is still to be desired from the standpoint of definite information and data concerning aphasia.

As Monakow stated (1906), it must be admitted that the more closely the clinical phenomena presented by patients with aphasia are studied, and the more accurately the anatomic changes are examined, the greater is the difficulty of furnishing a satisfactory explanation of the connection between the site of the lesion and the symptoms of aphasia in observed cases, and in spite of the persistence or increase in the size of the lesion in the speech region, the aphasic symptoms disappear; in other cases, the lesion may be beyond the limit of the speech region, yet the aphasia may persist for a long time. Paradoxical cases of sensory aphasia with destruction of Broca's area and motor aphasia with lesions in the temporal lobes are as yet unsatisfactorily explained. Monakow would explain many of the foregoing apparent discrepancies on the basis of a temporary suspension of function arising from the local interruption by disturbance of a tract of fibers which directs or carries out the function of a neighboring part. Monakow terms this diaschisis and states that aphasic symptoms are partly those of a more or less temporary character when not due to "neighboring symptoms" or to allied circulatory disturbances and are produced by diaschisis. The aphasia may depend on the diaschisis in distant regions of the brain more than on the lesion itself.

This patient, it is to be emphasized, was by birth and by nature left-handed. The right hemisphere, it is to be inferred, had become functionally developed. Furthermore, this condition developed in a person whose writing center was not brought into use until she learned to write, at least five years after the normal development of the cortex and pathways were completed; in other words, this function of the cortex, in this particular region, was acquired later than most of the coordinate motor functions.

It seems reasonable to conclude that a widespread lesion, as is evidently present in this case, if we are to judge from the total hemiplegia and hemianesthesia, may interfere with cerebral function as a whole (and I am assuming that we cannot restrict the functions of speech to a localizable motor region of the cortex). In fact, the effects of more or less localized lesions on the

entire cerebrum are by no means slight. In this case I believe that the agraphia on the otherwise unaffected side is dependent on the lesion at a distance. The function of writing, which in this case was acquired later than other motor functions, was less stable and therefore more easily affected, perhaps by diaschisis, but certainly by reason of its being comparatively a less organized function.

DISCUSSION

DR. CHARLES K. MILLS: No doubt in this case the right hemisphere primarily was the active engine in speech, writing and perhaps much else. The right hand was trained to do many things which the left hand otherwise would have done. The real ability to write remained in the right hemisphere in a congenitally left-handed patient, and when a large left-sided lesion of the brain occurred, although there had been an apparent transfer, it did not really result; the patient returned, so to speak, to congenital conditions.

DR. FRANCIS X. DERCUM: Dr. Buckley's case calls to mind one that was reported by Byrom Bramwell many years ago, in which the patient first suffered from a lesion of the left side of the brain, became aphasic, subsequently recovered the speech faculty, and then had a second lesion on the right side of the brain. He again became aphasic and remained aphasic permanently. Dr. Buckley's case, like Dr. Bramwell's case, is suggestive of a double rôle played by the two hemispheres in regard to the function of speech, although this function is one that is highly specialized. Dr. Buckley spoke of his patient as recognizing separate letters. She was notwithstanding unable to read, that is, she could not combine the separate letters into words or the words into phrases and sentences: in other words, she had alexia. One of the remarkable clinical facts of aphasia is that in so-called motor aphasia we always have alexia. I have met a motor aphasic who could read. Writing also is a highly specialized faculty and one acquired relatively late, and it would seem to me that the facts in regard to alexia apply also to agraphia.

I am very glad that Dr. Buckley clearly separates the purely dysarthric symptoms from the intellectual defects of aphasia. Further, I think we are too much in the habit of speaking of the specialization of the left hemisphere. The biologic fact is that the left hemisphere is always the larger of the two, it is dynamically the greater of the two and it almost stands to reason that when this greater half is crippled by a lesion, the functions of the right hemisphere are also impaired, that is, they are inhibited; and von Monakow's theory of diaschisis has a direct application here. The acquisition of left-handedness by right-handed persons is of course not unusual. We all of us recall the instance of a distinguished surgeon who was the teacher of many of us who was ambidextrous, and this quality had its origin in the fact that very early in life he suffered a serious injury of the right hand so that the left hand was of necessity trained, and trained very early.

DR. SPILLER: It is generally stated, and sufficient cases are on record to show the truth of the statement, that in right-handed people the speech area is in the left side of the brain, and it is probable that in left-handed people the speech area is in the right side of the brain. There are a few cases recorded in which, in right-handed people, the speech area has seemed to be on the right side of the brain, not on the left. There are cases recorded in which Broca's area on the left side has been destroyed, without disturbance of speech. It is important to determine whether the lesion in such cases developed early in life. It is unusual to find persistent aphasia in a right-handed

child who has a right hemiplegia that developed early in the left, because the right cerebral hemisphere is capable of assuming the speech function when a lesion develops within the first few years of life.

There are people who are ambidextrous, who probably have the speech mechanism in both sides of the brain. It is customary for a person who is left-handed to learn to write with the right hand. He is taught to do this in school.

On one occasion I had reason to believe that a certain man had an abscess of the brain in the left temporal lobe. He had no signs of sensory aphasia, and he was said to be right-handed because he was known to write with his right hand. He did have the abscess in the left temporal lobe, although he did not have sensory aphasia, but he was left-handed, and his child also was left-handed.

It is interesting to see how readily a child may become left-handed. I recall a baby of parents who were right-handed, and no left-handedness was in the family. The child had used the right hand like any right-handed child until this hand was burned. The bandage on the hand made the child use the left hand, and after recovery from the burn of the right hand the child was left-handed and had to be taught to use the right hand again. If he had been allowed to use the left hand, it is possible that the speech area would have developed in the right cerebral hemisphere.

We must assume in Dr. Buckley's patient that the speech mechanism was on the right side of the brain. Agraphia may occur in sensory as well as motor aphasia. I do not believe that if his patient had not been taught to use the right hand for writing she would have been able to write with the right hand after aphasia developed. The peripheral mechanism is under the control of the cortical mechanism. The ability to write with the right hand in his patient probably was controlled from the right hemisphere. A right-handed person with agraphia cannot write with the left hand.

A STUDY OF THE SPINAL ACCESSORY NERVES FROM A CASE OF BILATERAL ACQUIRED SPASMODIC TORTICOLLIS. DR. M. A. BYRNES.

A review of the literature on the pathology of the peripheral nerves in this disease revealed only one instance in which a similar study had been made. Ballance at the conclusion of his article on torticollis merely stated that the nerves showed nothing abnormal. Southam had, however, examined a portion of the spinal accessory nerve which had been previously subjected to stretching. This nerve showed slight pathologic changes which were, no doubt, due to injury in the earlier operation, and Kadner studied the intramuscular nerve fibers in congenital torticollis, but failed to find any marked changes.

In the case reported, a portion of each spinal accessory nerve and the posterior muscles on both sides of the neck were preserved in formaldehyd. The nerve was stained in cross and longitudinal sections by the following methods: hemalum and acid fuchsin, Weigert, van Gieson and phosphotungstic acid. The muscle tissue was stained with iron hematoxylin and hematoxylin and eosin. The Weigert preparation of the nerve showed almost total disintegration of myelin. In both the cross and longitudinal sections stained with phosphotungstic acid, there was definite fragmentation of the axis cylinders. The muscle tissue showed the changes which usually have been observed in previously studied cases of this affection. So far as I have been able to determine, this is the first instance in which changes have been described in

the peripheral nerves in acquired spasmodic torticollis. I do not feel, however, that any conclusion as to the etiology or significance of this pathologic finding can be deduced from this single observation, nor am I of the opinion that these changes discredit the existence of a mental torticollis, although it is my belief that in this particular case the condition was of organic origin.

DISCUSSION

DR. ALFRED GORDON: Several years ago, with Dr. Rowe, an aural surgeon, I investigated pathologically the subject of so-called neuralgia. We examined the extirpated nerves in several cases of facial neuralgia and found some degenerative changes.

Book Reviews

THE ELEMENTS OF SCIENTIFIC PSYCHOLOGY. By KNIGHT DUNLAP, Professor of Experimental Psychology in the Johns Hopkins University, Baltimore; Author of "Mysticism, Freudianism and Scientific Psychology," "Personal Beauty and Racial Betterment," Etc. Price, \$3.50. Pp. 368. Illustrated. St. Louis: The C. V. Mosby Co., 1922.

Professor Dunlap has selected the topics for his chapters with some conservatism. Sense Perception, Thought and Thought Content, The Bodily Mechanism, Reaction and Consciousness, Instinct and Habit, Development of Perception, Space Perception, The Thinking Process, Affective Experience, The Empirical Self or "Me"—these are headings which we expect to find in any present-day text that claims to be orthodox. Concerning these conventional topics, however, Dunlap has written opinions which in many cases depart far from orthodoxy. Sometimes these opinions are quite original. They are always stated clearly and concisely.

The discussions in this book are concerned almost exclusively with conscious activities. It is not strange, therefore, that the first chapters are devoted to sense perception rather than to types of movement or some other supposedly more dynamic subject. We do not mean to imply that Dunlap's systematic position leans toward the static and structural. While his major interest is in processes that are conscious, he looks on this consciousness as something that is present under specified conditions of organic reaction. Mind, to him, is a social functioning of cells in the process of adjustment to stimuli. To this extent he is definitely in accord with the biologic type of functional psychology.

In considering the methods of psychology, Dunlap shows a strong liking for and faith in experiment. Certain advocates of introspection, who are also advocates of experiment, may be disturbed when they read this author's comparison of the two methods. Introspection, in practice, "very often seems to come down to the observation of all content, so far as possible, which is present during any given period of time. This method is sharply opposed to the experimental method, which always aims to narrow the observation at any given time to a small group or detail of content, selected in advance for observation, withdrawing attention for the moment from all other content." (p. 333.)

An important element in Dunlap's general position is his assumption that consciousness, while empirical fact, is not observable. The observable facts of psychology are the somethings of which we are conscious. Often such presuppositions, though interesting in themselves, do not affect a writer's more specific views. But that is not the case here. When he comes to consider imagery, Dunlap is not confronted with the usual problems that arise in this connection. An image is something thought of, rather than a process of thinking. Since we can think of colors or sounds or tastes, it is not difficult to account for different modes of imagery. The modality of an image is identified with something thought of, and, since the thinking of is not itself observable, naturally it can have no modality to be explained. At least, this is the way Dunlap's position impresses the reviewer.

Dunlap's analysis reduces the experienced world to *sentienda* (sense qualities?) related to each other in intricate ways. It contains nothing else. Feelings are particular organic or 'bodily' *sentienda*. The relations existing between *sentienda* (or between other relations) are objects of consciousness. They can be perceived and thought about just as can *sentienda*. Some of the simpler relations are identity, difference and betweenness.

Dunlap thinks of habits as modifications of instinctive reactions. He criticizes McDougall and others for basing their classifications of human instincts on teleological considerations. In connection with affective experience, however, he proposes a theory of fundamental desires which does not differ markedly from McDougall's formulations.

An appendix to the book gives descriptions of certain abnormal conditions, that is, the psychoses, neuroses and amentias. This chapter is useful in itself, but it surely could be improved if the phenomena it describes were considered more explicitly in the light of habit formation and other fundamental concepts of functional psychology.

Taken as a whole, Dunlap's book is a worthy companion for the other serious texts in psychology. The style is straightforward and business-like. The student who works though it will not be amused, but, if he has caliber, he will be interested. There seems to be a general impression abroad that a textbook in psychology must be dogmatic. This one follows that rule to a considerable extent. Where the majority of facts or opinions supports a particular view, probably there is little danger in stating that view as *the view*. There are instances, however, in which Dunlap, like other authors subscribing to such a policy, states as fact opinions having scanty support. For instance, he describes the epicriticoprotopathic theory of cutaneous sensitivity as advocated by Head as established fact, whereas experimental evidence has rendered it extremely questionable. He has also stated the facts of color vision in terms of a theory—the three-color theory. Even granted that such a theory is the most satisfactory, how can the student of elementary scientific psychology be expected to keep a clear distinction between the theory and the facts? Much of the literature on color vision is needlessly difficult because of this practice of mixing fact and theory. As already indicated, however, such faults, to the extent to which they are faults, are shared with Dunlap by the run of writers of textbooks in psychology.

The publishers deserve credit for a fine piece of workmanship.

ANOMALE KINDER. VON DR. L. SCHOLZ. Dritte, Umgearbeitete Auflage von Prof. Dr. Adalbert Gregor, Direktor der Fürsorgeerziehungsanstalt Flehingen in Baden. Paper. Price, 72 marks. Pp. 312. Berlin: S. Karger, 1922.

As noted in the preface, this book commends itself especially to teachers of defectives and "special help" classes. It is written in popular style, explains the technical terms used and covers the subject matter in a satisfactory way. It should prove especially useful as a text for child-study clubs. The range of topics is indicated by the following chapter headings: Borderland of Mental Health; the Feeble-minded; Nervousness; Hysteria; Epilepsy; Chorea; the Psychopaths; Disturbances during Puberty; Suicide; Treatment and Prophylaxis.

The problem of abnormal behavior in children raises so many issues, none definitely settled, that each page is a challenge to the critical-minded. Devia-

tions from normal behavior can be roughly ascertained only providing a standard of behavior for the different ages has been established. The author stresses this point when he speaks about the gradations between normal and abnormal and defines the healthy child as one corresponding in his behavior to the standard for its age. But such standards have not been established, and the "percentages of deviation" productive of abnormalities have not been ascertained. The author is thus forced to the traditional manner of handling the subject, regarding for example "the nervous child" as full-bloom an entity as Kraepelin's dementia praecox. As in most popular treatises on this subject, there is much speculation and much propaganda—for prohibition and eugenics. Unusual stress is laid on alcohol as a cause of feeble-mindedness and epilepsy. The author states, as though they were well established facts, that one third of all epileptic patients have a direct heredity of alcoholism, and that psychopathic children, through alcoholic indulgence, can be made epileptic. In the same manner is the rôle of masturbation exaggerated in the etiology of insanity. In the chapter on the feeble-minded, the contributions of American clinical psychology appear to be unknown, nor are any references made to the literature on delinquent behavior. Tests of intelligence are cited without any reference to standards. The author regards intelligence tests as having only mass-value and finds them of little or no use in clinical diagnosis. This judgment of the clinician is based on the findings of physical stigmas, on the appearance of the patient ("Does he look bright or dull?"), on his history, especially in regard to spontaneity of interest, laughter, sense of humor, attention, response to pain and to a series of "gross" intelligence tests. This typical attitude of the clinician is maintained more or less throughout the subjects enumerated. Its failings are obviously in respect to the fine gradations which the author points out in his introduction, and which require a more refined method of approach than clinical deduction. In the chapter on treatment, the rôle of prophylaxis is considered especially in regard to social reform movements. Behavior problems as such are not considered. Delinquent and truant behavior, unlike sex aberrations, are all treated as symptoms of mental aberration. This precludes what has come to be regarded as an important chapter in abnormal behavior—not necessarily the behavior of abnormal children, but behavior byproducts of our social and economic structure. There is again no cognizance of the vast American literature on the subject.

In spite of the shortcomings inherent in the popular presentation of this subject, the book remains one of the best of its type.

SYPHILIS OF THE INNOCENT: A Study of the Social Effects of Syphilis on the Family and the Community, with 152 Illustrative Cases, Made Under a Grant from the United States Interdepartmental Social Hygiene Board, by HARRY C. SOLOMON, B.S., M.D., Chief of Therapeutic Research, Boston Psychopathic Hospital, Instructor in Psychiatry and Neuropathology, Harvard Medical School, and MAIDA HERMAN SOLOMON, A.B., B.S., Research Social Worker, Boston Psychopathic Hospital, Boston. Washington: United States Interdepartmental Social Hygiene Board, 1922.

In attempting to review a book, it is important, first, to know its purpose. This is an intensive study of the family and social importance of syphilis. It was carried out under a grant made by the Interdepartmental Social Hygiene Board. An attempt has been made to present the subject of syphilis

in its social aspects portraying the practical problems as they actually arise, and also to show that they are of more than purely medical interest. The subject is treated, first, in relation to the individual; second, as concerning the mate; third, as affecting the child; fourth, as influencing the family, and fifth, in relation to the community.

The book is fully documented, and the study is based largely on observations made at the Boston Psychopathic Hospital and in that community. The old idea that an immune woman might give birth to a syphilitic child, under what has been called Colles' law, is conclusively shown to be without foundation; as is also Profeta's law that an apparently healthy child of a syphilitic mother could be nursed by its mother or a syphilitic wet nurse and yet not be infected. The employment of serologic tests has finally settled these questions.

The authors believe that there are different strains of the syphilitic organism, and they produce some case histories to support that contention.

Very properly, stress is placed on the necessity of making a careful family examination in any instance in which one member has been infected; the authors have found less difficulty in carrying out this investigation than is ordinarily apprehended by the medical practitioner. The authors properly attach more value to the clinical examination than to the result of the Wassermann test taken by itself. The question of the influence of syphilis on marriage is discussed at considerable length, without, however, reaching conclusions that are entirely definite. In the same way, legislation looking toward medical certificates of freedom from venereal disease is also set forth, with general approval of the intent of such law and full recognition of the difficulties of applying them, and with some suggestions for modifying them in the direction of uniformity.

Finally, the question of syphilis in the community is taken up from the financial standpoint, and an attempt is made to indicate in some degree the cost to the community of this disabling disease.

The book is prepared so as to be readily understood by the lay reader, but it gives such a general view of the disease in its medical and social relations that it is well worth reading by every medical man and every social worker.

THE PSYCHIC HEALTH OF JESUS. By WALTER E. BUNDY, Ph. D. Pp. 299. New York: Macmillan Co., 1922.

The author, associate professor of the English Bible in De Pauw University, sets forth to prove that the psychiatrists and neurologists who have written estimates of the mind of Jesus have held perverted views. An analysis of the "pathographers" whose views regarding the mental state of Jesus are cited, reveals no name of importance in the field of psychiatry. One is inclined, indeed, to doubt that any scientific psychiatrist would be greatly interested in the analysis of the writings attributed to Jesus for the purpose of forming an opinion as to his mental state. True, Freud and his disciples have read out of the lives of many great artists, dramatists and writers queer conclusions as to their sexual habits which do not seem to be borne out by any actual evidence; it might, indeed, be better to state that they have read into these writings more than the authors ever put there. But the problem of Jesus is a far more complicated matter. It may safely be stated that no man can approach the subject with the cold abstraction that scientific analysis demands; the authorities

whom Professor Bundy attacks so vigorously were not scientific psychiatrists but impassioned philosophers with some knowledge of psychology and diseases of the mind; and all of the men who oppose them, including Professor Bundy, were theologians, that is to say, believers whom one must convince against their will. Except as forms of mental exercise, such studies are as fruitless as the puzzles of Sam Lloyd. The modern psychiatrist will find a sufficient number of problems awaiting him in the etiology and treatment of mental diseases which continue to attack hundreds of thousand of living human beings in 1922. The essays on the psychology and mental health of the prophets of old may well be left to the theologians.

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GENESIS OF CEREBELLAR FUNCTIONS

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In this discussion of the cerebellum, an attempt is made to visualize the genesis of cerebellar functions.

Realizing how far in the future definitive conclusions in this subject must be, I prefer to consider this communication as a study embodying certain of my present views, leaving my position still flexible and open to whatever modifications further investigations may dictate.

Many difficulties have previously been encountered in reaching a solution concerning cerebellar functions. Indeed, it seems almost impossible to draw any comprehensive deductions regarding this question from all of the evidence at present available. Animal experimentation and clinicopathologic observation in mammals and man leave much to be desired. A vast amount of experimental work has been carried on without the necessary preceding morphologic understanding of the cerebellum, and the results, in consequence, have often confused rather than clarified the situation. By far the majority of workers in the past have directed their attention to the organ at a stage in its development when it has reached its highest complexity. This necessarily has imposed all of the difficulties of approaching the problem in its most complex rather than in its simplest form.

It is my intention to pick up the threads in the genesis of this organ where they may be traced separately in the most primitive forms of vertebrate life, before they have woven themselves inextricably into the complex fabric of the ultimate cerebellum. Starting from this, its organic inception, it may be easier to follow step by step the progressive evolution of structure which has gone hand in hand with the advancing development of motor control as the vertebrates acquired new capacities and adjustments.

The citation of authorities on this subject is a matter so familiar that it seems unnecessary to include here the various opinions formerly expressed. The reader, however, is especially referred to the works of André-Thomas, Babinski and Tournay, Luciani, Bechterew, Bârâny, Bolk, Van Rynberg, Mills and Weisenburg.

I wish to offer a word of apology in connection with the nomenclature. In order to place the cerebellum in proper alinement for the description of certain evolutionary phases in its genesis, it has seemed necessary to select for it the term *parencephalon*. This brings the terminology into more precise consonance with that of the other brain vesicles, such as *diencephalon* and *mesencephalon*. Although the addition of another term to an already overburdened vocabulary is perhaps unfortunate, this one is selected advisedly, because it was employed by Aristotle, who called the cerebellum "*parencephalis*."

The method of approach in this investigation includes comparative morphologic studies beginning with the more primitive forms of vertebrate organization, such as cyclostomes, sharks and rays, ganoid and teleost fish; with later reference to reptiles, amphibians, birds and mammals. The embryogenetic characters of the cerebellum were studied in all of these forms with the idea of revealing a common ground-plan in the structure of the organ, together with an analysis of the motor reactions of the animal in the light of its cerebellar organization. Some preliminary experimental work was done on sharks, pigeons and cats.

The problem of this research may be phrased in three queries: 1. When, in the course of animal organization, did the cerebellum make its first appearance? 2. What were the probable morphologic and physiologic conditions which established the demand for this organ, and what evidence is there that the cerebellum in its primordial form became essential to the posture-maintaining quality of motor control? 3. In what manner did the cerebellum, once having acquired its specialization for dominating the posture-maintaining elements of motor control, eventually establish within itself a hierarchy regulating all of the further expansions and modifications of this quality of motor activity?

THE FIRST APPEARANCE OF THE CEREBELLUM

The first query may be answered categorically in the statement that no exact homologue of the vertebrate cerebellum is known in the invertebrate phylum. The cerebellum, therefore, made its appearance at some critical period during the invertebrovertebrate transition in response to certain functional necessities which did not exist in the invertebrate.

It might appear unwarranted, however, to assume so dogmatic a position without first considering some of the conditions of invertebrate life. Insects, for example, must utilize some stabilizing and balancing mechanism for the delicate adjustments of their bodies in flight through the air, in alighting or in starting to fly. Furthermore, such invertebrates as move about in the water, employing various means of locomotion, must likewise be in need of a definite equilibratory apparatus, and this also applies, perhaps in a less degree, to land-living insects or other invertebrates which crawl or creep.

In the king crab (*Limulus polyphemus*), Patten¹ found by experiment that there is a part of the central nervous system the destruction of which produces a marked disturbance in the animal's equilibrium. After cutting or removing the vagal neuromeres, it is impossible for the animal to right itself if placed on its back in the water. If the experiment is confined to one side, the crab constantly moves in a circle to the side of the operation. The circular movements continue for hours at a time. Incision or extirpation involving any part of the brain behind or in front of these vagal neuromeres does not destroy the tendency or power of the animal to right itself or properly direct its locomotion when able to move.

Patten¹ is of the opinion that the subdivision of the incipient vertebrate head is to be observed in the arachnids, and that the brain vesicles follow a plan of subdivision analogous to that in vertebrates. The fourth brain vesicle in arachnids is the metencephalon or vagus organ. The lateral stomodeal ganglion is connected with this part of the brain and is united by a large commissure forming a prominent arch over the neural surface of the stomodeum. It is gradually crowded caudad by the migration of the mouth and rostrum, as well as by the increasing size of the lateral eyes, forming in vertebrates the rudiment of the cerebellum.

Although Patten¹ believes that the median stomodeal ganglion of arthropods and the cerebellum of vertebrates are the only brain structures that arise originally in the median line, it is by no means clear that this median stomodeal ganglion is in any sense the homologue of the vertebrate parencephalon. Indeed, there is much doubt in the light of the embryogenetic process which results in the formation of the cerebellum in the lowest vertebrates, whether this median stomodeal ganglion is in any sense homologous with the parencephalon. The mode of development of the cerebellum in the cyclostomes and in all the true fish points more conclusively to a bilateral paired anlage than to a median unpaired mode of derivation.

So for the present at least, and until much more extensive studies in vertebro-invertebrate homology may be completed, it is fair to assume that the vertebrate cerebellum has no exact counterpart in the invertebrate phylum, and that this organ should be regarded as a distinctive vertebrate character. It came into existence during an epoch-making period when a critical transition led to the vertebrate differentiation out of some invertebrate line. Thereafter it has maintained itself with increasing prominence as one of the chief features in the vertebrate brain. Even thus interpreted, it must still remain a question whether

1. Patten, W.: The Evolution of the Vertebrates and Their Kin, Philadelphia, 1912.

the cerebellum is a neomorph. It is, perhaps, more far-seeing to regard its fundamentals as already laid down in some diffuse invertebrate rudiments ultimately assembled as a distinctive vertebrate organ under the influence which determined the inception of the vertebrate phylum.

THE MORPHOLOGIC AND PHYSIOLOGIC CONDITIONS LEADING TO THE DEVELOPMENT OF THE CEREBELLUM

Consideration of the next phase of the problem deals with the probable morphologic and physiologic conditions which established the demand for this organ. This opens directly into one of the most fertile fields of evolutionary research, namely, the mode of transition from the invertebrate to the vertebrate.

The evidence furnished by living and fossil forms is by no means sufficient to be entirely convincing concerning the nature of this process. In consequence, a number of theories have been advanced and have had earnest advocates, so that the subject is still a matter for debate. But whatever divergence of opinion may exist with reference to this

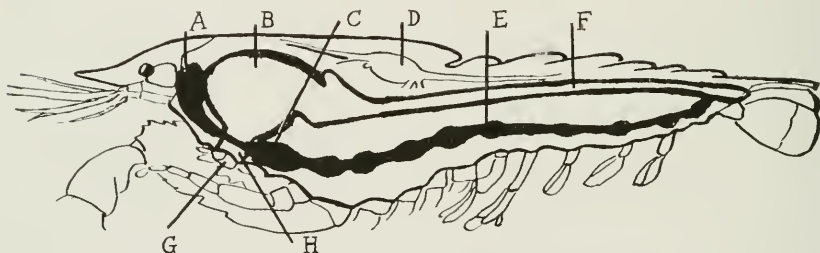


Fig. 1.—Midsagittal section of lobster (*Homarus americanus*) showing relation of the stomach and nervous system. *A* indicates supra-esophageal ganglion; *B*, stomach; *C*, infra-esophageal ganglion; *D*, heart; *E*, ganglionated cord; *F*, intestine; *G*, mouth; *H*, esophagus.

evolutional process, there is real unanimity concerning the most outstanding difference between the organization of the higher segmented invertebrate and that of the vertebrate. This difference depends on the position of the mouth, together with the relation of the alimentary canal to the nervous system, which relation the position of the mouth appears to determine (Fig. 1).

In the higher segmented invertebrates (arthropods, such as the king crab, lobster or cray-fish) the mouth occupies a position ventral to the lateral eyes and is continuous with a slender tube, the esophagus. This tube passes between the head ganglions to the stomach which lies as a dilated pouch dorsal to the brain. To all intents and purposes, therefore, the esophagus passes through the center of the brain, being surrounded on all sides by central nervous tissue.

The caudal prolongation of the nervous system extends toward the tail from the infra-esophageal ganglion, and lies ventral to the intestinal

canal. By this arrangement the entire nervous system occupies a position in front of the alimentary canal, which relation is exactly the reverse of the enteroneural relation in the vertebrates. Although this relation of the stomach and intestine to the central nervous system and the general configuration of the body may have certain mechanical advantages in aiding the animal to maintain the position best suited to its purposes of life, it is subject to one serious disadvantage. The esophagus being surrounded by the brain, at once becomes jeopardized as the conducting tube from the mouth to the stomach should the brain for any reason take on increased growth. By such growth in the brain the esophagus would become more and more constricted until reduced to the smallest dimensions, as it is in many insects which are for this reason forced to depend on fluid alimentation sucked through the narrow tubular esophagus into the stomach. Any evolutionary development in the brain, therefore, by reducing the caliber of the esophagus, would threaten the whole process of food ingestion, and thus have profound effects on the metabolism of the animal.

This compromising relation of the esophagus to the brain which occurs in the invertebrate is completely changed in the vertebrate, for in such forms of animal life the stomach and intestine have assumed a position ventral to the nervous system, and the esophagus is no longer embarrassed by the necessity of passing through the brain in order to reach the stomach. Such a great and far-reaching alteration as this in the neuro-enteric relation has provided the brain with an opportunity to expand as it may without jeopardizing the alimentary canal or exacting a limitation in the food supply of the animal.

The perplexing question is: What were the influences and processes which produced this alteration in the relation of the nervous system to the digestive tube?

Geoffrey St. Hilaire² advanced the explanation that this great change was wrought by the simple process of the invertebrate turning over on its back and swimming in this position, thus gradually converting its dorsal into its ventral surface and causing the appearance of a new mouth, which opened in this case on the new ventral surface directly in the alimentary canal without passing into the brain, while the old mouth in the dorsal position became obliterated.

Another view concerning this epoch-making alteration of relations was advanced by Bateson,³ who maintained that from the beginning there were two types of bilateral elongated segmented animals which differentiated along two distinctly different lines. In one the alimentary

2. St. Hilaire, G.: *Sur la vertèbre*, La rev. encyclopedique, 1822.

3. Bateson: *The Ancestry of the Chordata*, Quart. J. Micr. Sc., 1886, vol. 26. *Materials for the Study of Variation*, London, 1894.

canal was dorsal to the nervous system, and these animals in their highest evolutionary form became the arthropods; while in the second, the nervous system was dorsal to the digestive tube from the beginning and reached the final evolutionary stage in the vertebrates. The latter view, interesting as it may be, lacks the necessary evidence to establish these two distinctly different types of ancestral animal life, either living or fossil.

Among the most suggestive theories explaining the manner in which the nervous system assumed a relation dorsal to the gut tract in the vertebrate is the one proposed by Gaskell.⁴ He recognized the necessity imposed by the invertebrovertebrate transition for a rearrangement of the relations in the neuraxis and the digestive tract, and he believes that this took place by a progressive invasion of the walls of the stomach by nerve cells lying in the brain ganglions in the invertebrate. Little by little this invasion of the pouch by neural elements converted the walls of the stomach into a solid mass of nerve tissue on either side, surrounding the old epithelial lined cavity of the stomach, which now becomes the ventricle of the brain. This invasion of the stomach is not complete, for in the region of its roof it still retains its old epithelial character even where the invasion of the nerve cells attains its most extensive representation in the brain of the higher mammals. Here, in this region of the roof, the epithelial lining of the old stomach is thrown into many glandular reduplications represented by actual glandular tissues in the different orders of vertebrates, thus showing the potentiality of the roof of the brain to manifest its old gastric proclivities in the formation of glandular structures. These glandular structures include the paraphysis and the dorsal sac, the choroid plexuses and the pineal gland.

After the invasion of the stomach by these nerve cells coming from the ventral brain ganglions of the invertebrate, the nerve cells from the ganglionated cord follow suit, invading the floor and side walls of the intestinal canal, thus forming the spinal cord whose central canal in the vertebrate represents the primitive invertebrate channel of the gut.

Gaskell⁴ finds substantiation for this view in many facts related to the central nervous system, but the one of cardinal importance to him is the position of the infundibulum which in the embryo passes from the region of the mouth cavity into the third ventricle. This tube, lined with epithelium, is a remnant of the old invertebrate esophagus, and the fact that it has become invested by nerve tissue is simply another illustration of the neural invasion from the head ganglions. According to this view of Gaskell, it is not necessary for the animal to turn over and swim on its back. All it has to do is to

4. Gaskell, W. H.: *The Origin of Vertebrates*, London, 1908.

sacrifice a perfectly good stomach in the interests of forming a better brain, and also to give up an efficient gut tract in order that it may win a spinal cord.

This supposed critical exchange between brain and stomach probably points to the most glaring defect of Gaskell's theory, for if the invertebrate thus transformed its gut tract into a central nervous system, what was left to it for the purposes of alimentation? It may be in the line of efficient evolutionary progress to produce a more highly developed nervous system, but a brain without a stomach to sustain it would bear little of promise to the animal.

Patten,¹ although in accord with Gaskell in certain main lines of the process which resulted in the changed neuro-enteric relation, differs materially with reference to the manner of this change. He, like Gaskell, believes that the line of ascent was through the arthropods and thence carried on by the ostracoderms, a class of animal midway between the most primitive vertebrates and the highest segmented invertebrates. These animals are now entirely extinct and seem to have been present only from the Silurian to the upper Devonian age.

Patten's interpretation of this transition draws on the imagination much less than does that of Gaskell. He recognizes the necessity of providing a new entrance into the gut tract in order to escape the constricting influence of brain growth on the esophagus. This, he believes, is accomplished by a gradual shifting of the old mouth from the ventral surface of the head into what, for the invertebrate, would be a dorsal position. As this gradual shifting took place, a mouth was established which provided a direct connection between the esophagus and the stomach, without passing through the brain. According to Patten's view, this relatively simple shifting of the mouth would then convert the ventral invertebrate surface into the dorsal vertebrate aspect of the body and determine a gut tract ventral to the central nervous system. The opportunity for developing such a new mouth was provided, according to Patten, by the presence of a very ancient organ in the arthropod head, known as the cephalic navel or dorsal organ. This stood ready to take the place of the old mouth which was slowly being eliminated. "Its presence alone made the existence of the vertebrates, as we know them, a possibility."

The development of a vertebral endoskeleton, according to most authorities, does not seem nearly so difficult to explain as this profoundly important relation of the nervous system to the digestive tube. When it was finally determined with the definitive arrival of the vertebrate, by whatever evolutionary process it may be, animal organization had come into possession of an entirely new physical proposition. The stomach and intestine, which in the invertebrate occupied a position in

the back, in the vertebrate now occupied a position in the belly. Mechanically, this new relation of the central axis of the vertebrate to the gut and stomach imposed the necessity for new functions in maintenance of the best posture in which the animal might live. The intestine and stomach, filled as they were with the gaseous contents of the alimentary canal, made the ventral aspect of the body lighter, and unless some means were devised to hold the belly down, the animal would be turned over on its back and thus be deprived of its best opportunities for dealing with the elements of its environment. In order, therefore, to maintain what may be called the optimum physiologic posture, there arose the necessity for an organ which would so synergize the ventral, dorsal and lateral musculature as to hold this position.

The earliest vertebrates of which there is evidence, namely, the cyclostomes, comprise a group of rather sluggish water-living animals. The best known representatives of this group are the lampreys and the hagfish. These animals present all of the characteristic vertebrate features. They have a well demarcated head and a long segmented body, gills and a gut tract, which lie ventral to the brain and spinal cord. The mouth is large and circular, without jaws, and equipped with rough, small, stonelike teeth. These animals have a well marked dorsal fin and an inconspicuous ventral fin, but no paired lateral fins or other body appendages. In another important respect they differ from the invertebrates, i. e., in the possession of a series of organs situated along the lateral aspect of the body, on either side, connected with the brain, known as the lateral line organs, which function in the acts of balancing. In addition to these organs of balance, the cyclostomes have two semicircular canals in the head which are exquisitely sensitive to changes in position and are important structures in the maintenance of equilibrium.

Invertebrates appear to have no organs homologous to the semicircular canals. Some insects have specialized auditory organs which probably are more closely related to the sense of hearing than to the function of balance. Certain hairs scattered over the body of some insects and on the antennae are sensitive to sound and may be related to the sense of balance. The basal joint of the antennae of some insects forms a socket richly supplied with sense organs containing a large ganglion supposed to have a functional responsibility related to equilibrium. Nothing comparable to the semicircular canals, however, which made their first appearance in the cyclostomes, has been observed in invertebrates.

The life history of one of these primitive vertebrates sheds much light on the mechanical handicap imposed by the now ventrally placed alimentary canal (Fig. 2). Selecting as an example the lamprey (*Petromyzon*), it is found that after leaving the egg the young must

shift for themselves. At this period the animal is known as the ammocoete, or larval form of *Petromyzon*. For the first three years of its life, the ammocoete lies submerged in the mud in shallow waters. During this time it depends for its nutrition on what is brought to it by the moving currents of water above it. It lies flat on its belly with its dorsal fin directed upward. It has little independent motor life, except those slight activities necessary to maintain an optimum physiologic position. Its mouth is directed forward, while the position of the body is best calculated to facilitate its bilateral symmetrical growth. At this stage the nervous organization is relatively simple, although it contains all of the fundamentals of the ultimate vertebrate neuraxis. Its cerebellum consists of a heavy ridge of nerve cells projecting from either side into the fourth ventricle and part of the alar plate of the afterbrain. Because of its constant relation throughout the entire vertebrate phylum, and also because of its position in the bulb, it is here called the cerebellum bulbare. Through the eighth cranial nerve it receives a large collection of fibers coming directly from the semi-

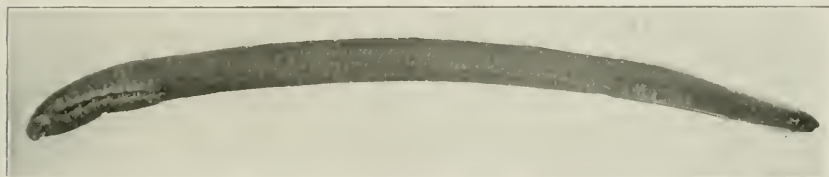


Fig. 2.—Ammocoetes, larval form of petromyzon as it is during the period when it remains embedded in the mud.

circular canals. Some fibers also enter it from the lateral line organs, and probably some—although this point has not been determined with full satisfaction—from the muscle plates. At this early stage, therefore, the ammocoete maintains its optimum physiologic position in response to afferent impulses which come from the extremely sensitive semi-circular canals, from the lateral line organs and probably from the muscles. These impulses enter the bulbar cerebellum and are then transmitted to the effectors for maintenance of equilibrium. The entire cerebellum at this period is represented by two long ridges of nerve cells, one in either wall of the fourth ventricle.

At the end of three or four years, the ammocoete emerges from its mud encasement and becomes a free-swimming fish. It changes at this period from its larval to its adult form, and during this metamorphosis acquires its buccal funnel and its curious rows of stonelike teeth in its circular mouth. The eyes, which hitherto have been atrophic, now approach the surface of the head and become functionally active. The endostylar groove is converted into the thyroid gland, and almost

immediately thereafter the animal manifests the tendencies of its parasitic life, attaching itself by means of its suckorial mouth to some other fish which it proceeds to eviscerate. At this period when the animal requires more control of its motor activities, the two long, lateral ridges in the side walls of the fourth ventricle increase rapidly in size. At their cephalic extremities a well defined bar of cells with nerve fibers connects these two ridges across the median line above the floor of the fourth ventricle (Fig. 3).

This connecting piece which joins the two lateral ridges is called the cerebellum jugale. It makes provision for the correlation of movements on the two sides of the body. Its important physiologic significance lies in the fact that it produces the synthesis of afferent nerve impulses necessary to bilateral synergized action of the body. This structure, the cerebellum jugale, I believe, foreshadows the development of the inferior vermis in the ultimate cerebellum.

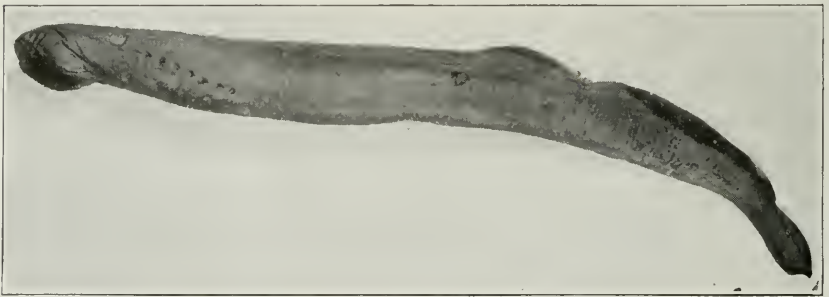


Fig. 3.—Adult *Petromyzon* showing development of circular mouth and dorsal fins. There are no lateral appendages.

From the evolutionary standpoint, therefore, the organ representing the most primitive cerebellum, so far as we know, may be called the archeparencephalon (original or primitive cerebellum). It occurs in cyclostomes, including the lampreys (*Petromyzonidae*), hagfish (*Myxiniidae*) and bdellostoma. It comprises two morphologic structures, the cerebellum bulbare, represented by a ridge in either lateral wall of the fourth ventricle, and a connecting or yoke piece projecting into the ventricle, the cerebellum jugale.

These facts are illustrated in Figure 4, a reconstruction of the adult petromyzon brain shown in midsagittal view. In its genesis, the cerebellum at first shows no evidence of coalescence across the midline by means of a supraventricular connecting piece. During the entire period of larval life, while the animal is lying quiescent in the mud, the cerebellar structure consists of the two unconnected lateral ridges, constituting the cerebellum bulbare. As it approaches metamorphosis,

however, a strand of nerve cells begins to migrate across the roof plate. By the time the animal becomes a free-swimming organism, it has acquired a cerebellum jugale. From its inception, the cerebellum bulbare is connected by a massive group of fibers with the semicircular canals. Some fibers also enter it from the lateral line organs. These facts, taken in conjunction with the mode of life during the larval period, would seem to indicate that the cerebellum bulbare acts as a correlating organ for impulses coming in from the right and left sides of the body, through the receptors of the semicircular canals and lateral line organs, in order to maintain the animal in a posture best suited to its progressive development.

If left to the influence of physical forces alone, the ammocoete would undoubtedly be turned over on its side, or might be forced into a position resting on its back. Either of these postures would be disadvantageous

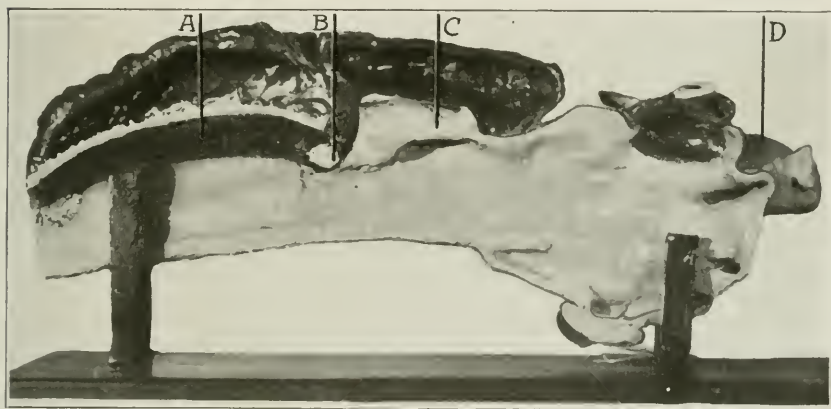


Fig. 4.—Reconstruction showing in midsagittal view the brain of *Petromyzon*. This specimen illustrates the position of the archeparencephalon (cerebellum bulbare and cerebellum jugale). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, mesencephalon; *D*, telencephalon.

in the obtaining of a food supply, or might even lead to the development of asymmetries and irregularities in the body formation. Chiefly in the interest of alimentation, then, the belly-down posture during the larval period must be accounted the best position for the animal, and this position is maintained by the mechanism provided in the presence of the lateral line organs and semicircular canals, mediating their motor effects through the cerebellum bulbare. This position of the animal which best meets its needs of life may properly be designated the optimum physiologic posture.

Immediately, however, on leaving its larval period and becoming a free-swimming fish, the animal is confronted with new problems. It

now has the more plastic environment of the water in which it must maintain its optimum physiologic posture. This posture is still that of the belly-down position, both in rest and in motion. It would now require the closer cooperation of the two sides of the body, and at this period, therefore, in response to this need, the cerebellum jugale probably made its appearance, connecting the two lateral ridges of the cerebellum bulbare across the midline.

The cerebellum, thus constituted, appears to be related to the most primitive portion of the motion-producing apparatus; namely, the archekinetic system, a mechanism which depends on relatively simple associated reflex arcs, and which is sufficient for the production of the animal's extremely limited range of movements.

In its histogenesis, the archeparencephalon, as studied in the ammocoete and petromyzon, shows a tendency of the cells aggregated in the dorsal portion of the alar plate to become more and more definitely isolated as a special localized area in the medulla into which enter the fibers from the semicircular canals and other proprioceptive organs of the body. From the definite connection of this part of the neuraxis with these proprioceptors, more particularly the semicircular canals, the inference seems justified that this region of the brain is primarily involved in some function pertaining to the proper maintenance of posture. This inference requires much further substantiation by means of experiment and other studies, but it is offered as the best hypothesis to be advanced on the evidence now in hand.

Histologically, the differentiation which takes place in these localized areas of the bulb forming the cerebellum bulbare and the cerebellum jugale, shows a tendency of a certain number of cells to assume large proportions and to be scattered diffusely in a dense matrix of smaller cells. No definite Purkinje cells or layer of cells may be discerned in *Petromyzon*. In fact, the differentiation into layers which is the characteristic feature of the cerebellum in all orders of vertebrates, does not appear distinctly in any of the cyclostomes. The tendency toward this stratification, however, is far enough advanced to foreshadow the ultimate arrangement of cellular elements in the vertebrate cerebellum (Fig. 5).

In concluding the consideration of the probable morphologic and physiologic conditions which established a demand for the cerebellum, the facts clearly point to the new mechanical condition impressed on the vertebrate by the alteration in the relation of the nervous system and the digestive tube as compared with the invertebrate. This new relation required a definite motor mechanism to maintain the optimum physiologic posture. The mechanism had its peripheral representation in the development of the lateral line organs and the semicircular canals, and its central representation in the archeparencephalon.

THE CEREBELLUM AS A POSTURE-MAINTAINING ORGAN

Accepting this conception of the cerebellum as an organ which differentiated for the primary purpose of maintaining posture, it is not difficult to perceive how, by expansion and accession, it took on itself the posture-maintaining function in all activities to which posture is essential.

Our idea of what posture is has been materially extended by the thought and researches of the last few years. Posture is not merely a requisite in the maintenance of the optimum position. Posture must be maintained through all motor action, for motion is actually a steady stream of fluid postures, just as moving pictures are produced by a steady stream of serial poses. In some previous work of the writer,



Fig. 5.—Cross section through the bulb of *Petromyzon* showing cerebellum bulbare (archeparencephalon). *A* indicates the cerebellum bulbare; *B*, the eighth nerve.

in collaboration with Dr. S. Philip Goodhart,⁵ it was clearly shown by means of the ultrarapid moving picture camera that every motion is composed of a consecutive series of postures which flow one into another as the movement is produced. The analysis of each movement demonstrates that the action begins from an initial posture and ends in a terminal posture. Between these two limits is a complete series of intermediate postures. A movement may be arrested at any point in

5. Goodhart, S. P., and Tilney, F.: Analysis of Somatic Motor Disturbances. Analysis of Motor Disorders by the Aid of Ultra-rapid Moving Pictures. *Neurol. Bull.*, **3**, Sept.-Oct., 1921.

its production between its inception and its termination, with the result that the part involved in the performance will manifest one of the component postures whose summation constitutes the act in question. Thus, raising the arm to point with the finger at some distant object, is an act comprising a complete series of postures, beginning with an initial stage, running one into another until the motion is produced and the terminal stage reached. All of the postures between the initial and terminal phases constitute a steady succession, flowing in the most direct manner to the accomplishment of the purpose. If the muscles producing this movement did not cooperate properly one with another, if the flexor groups were not properly adjusted to the extensor groups, distortions would occur in the motion and its production would not be the most direct or perfect execution from start to finish.

The matter of posture, therefore, is equally as important to action as it is to rest. Ramsay Hunt⁶ has made the statement that posture follows motion like its shadow; and as a pioneer conception this figure is convenient and welcome. But posture is much more intimately concerned with motion than even this would imply. It is incorporated within the very substance of motor activity and is not in any strict sense its mere shadow.

The motion program which embodies the purposes and designs the execution of motor performance, it is generally accepted, has its inception in the forebrain; while the posture pattern necessary to such a movement arises in the cerebellum. These two streams of motor innervation must run concurrently if motor performances are to be most direct and effective in their execution.

When motor activity became more and more complex, as for example, in passing to the next higher order of vertebrates—the selachians—there appeared in such forms as the sharks and rays a much greater capability of motor performance than in the simpler cyclostomes. The motor capacity of the sharks gave rise to much more extended and much more complex movements. Their great speed in swimming, their capabilities in pursuit and attack of prey, their ability to cruise about in the water for great distances, as well as the remarkable speed with which they may rise to the surface or propel themselves to the depths, all stand out in marked contrast to the limited and relatively simple motor performances of the lampreys and hagfish. This greatly increased motor capacity is clearly indicated in the expansion of the entire central nervous system, but nowhere is it more clearly emphasized than in the development of the cerebellum. That organ has obviously kept pace with all of the new accessions of motor activity

6. Hunt, J. R.: The Statesthetic and Kinesthetic Components of the Afferent System, *Arch. Neurol. & Psychiat.* **8**:311 (Sept.) 1922.

seen in the sharks and rays. Complex automatic associated movements of paired lateral fins, of dorsal fins and body segments with the tail, make possible a wide increase of motor performances, not only in the selachians, but in all true fish.

The posture-maintaining organ, in adjusting itself to this increased complexity of motion, has developed a new part of the cerebellum which does not appear in the lower vertebrates. Figure 6 illustrates the

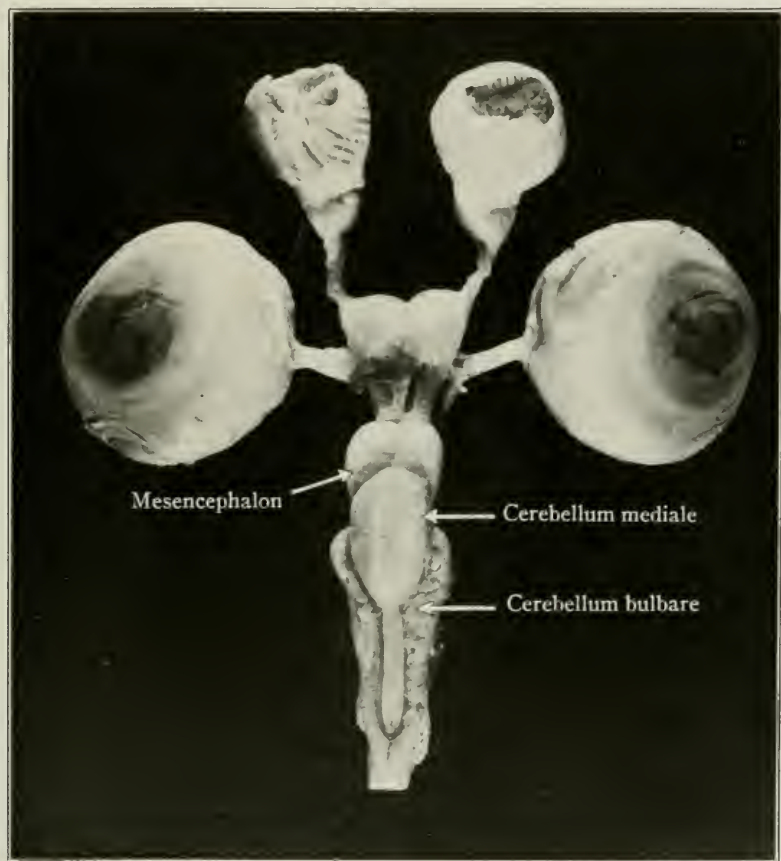


Fig. 6.—Dissection of an adult dog fish brain with eyes and olfactory organs attached, showing cerebellum bulbare and cerebellum mediale.

appearance of this new cerebellar element in one of the dog-fish (*Squalus acanthis*). It is a prominent accession to the cerebellar structures. The more primitive cerebellum bulbare may be observed much expanded in all its dimensions on either side of the medulla and extending forward toward the mesencephalon. It still occupies a prominent position in the dorsolateral wall of the medulla, and is even more

conspicuous than in *Petromyzon*. The cerebellum jugale connects the cephalic extremities of the two lateral ridges forming the cerebellum bulbare; it is now concealed by the superposition of the new cerebellar element which is here called the cerebellum mediale.

A cross-section of the dog-fish brain is shown in Figure 7. In it will be seen the new accession to cerebellar organization, the cerebellum mediale, lying ventral to which is the cerebellum jugale and flanking it on either side the two lateral ridges constituting the cerebellum bulbare.

Nothing resembling the cerebellum mediale is to be found in the lowest of the vertebrates, namely, the cyclostomes.

It is worthy of note that the cerebellum bulbare, as well as the cerebellum jugale, in all of the dog-fish has undergone a considerable expansion. Both of these parts of the primitive cerebellum have increased in size and complexity. Both now contain a granular and molecular layer. Large Purkinje-like cells are already present, arranged

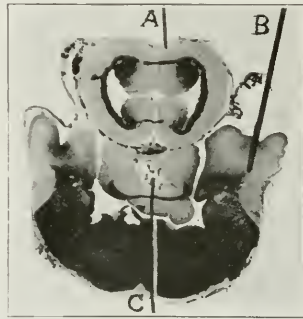


Fig. 7.—Cross section of the cerebellum in the dog fish. *A* indicates cerebellum mediale; *B*, cerebellum bulbare; *C*, cerebellum jugale.

in irregular rows on the boundary line between the granular and molecular strata. The reason for this marked increase and extensive differentiation in the archeparencephalon is undoubtedly to be found in the fact that the semicircular canal apparatus has greatly increased in size and complexity. Not only has a third semicircular canal made its appearance, but a complex appendage to the vestibular mechanism—the ductus endolymphaticus—has also developed. This addition to the labyrinth consists of a long tubular structure connected centrally with the atrium of the otocyst and extending to the surface of the head, where it becomes much convoluted and separated from the exterior by a relatively thin membrane. The ductus endolymphaticus appears embryologically in all vertebrates above and including the selachians, but in none does it reach this marked degree of complexity in its development. In the sharks it appears to be an organ closely related with the equilibrating apparatus, and from the position of its distal

extremity would seem easily accessible to the influences of pressure, depending on the depth of the water in which the animal swims. Its appearance in this complex form in the sharks undoubtedly explains in very large measure the pronounced differentiation occurring in the cerebellum bulbare.

There seems to be no reason to believe, on the grounds of anatomic evidence, that this increased vestibular complexity is in any way responsible for the appearance of the new element in selachians, namely, the cerebellum mediale. This structure is quite independent of any connection with the semicircular canals or lateral line organs. The fifth, eighth and tenth nerves, which bring in most of the fibers from the proprioceptive organs of the lateral line and semicircular canals, enter directly into the cerebellum bulbare, and many of them are seen streaming across the midline to terminate in the cerebellum jugale. None of these fibers has communication with the cerebellum mediale. From this fact, it is concluded that some reason other than the amplification in the equilibratory mechanism must explain the appearance of the new cerebellar structure. Its histology is similar to that of the cerebellum bulbare and the cerebellum jugale. It consists of a molecular and granular layer and contains cellular elements which in their position and character may be regarded as representatives of Purkinje cells. The cerebellum mediale encloses a large dorsal extension of the fourth ventricle, having a cavity of its own beneath whose ependymal lining run the heavy masses of fibers entering and leaving the organ. The connections established by these fibers, however, so far as they may be traced, indicate that this cerebellum mediale receives afferent impulses from the spinal cord, and in addition has an extensive communication with the midbrain and basal portion of the forebrain.

First inspection of a shark's brain might give the impression that the cerebellum was entirely represented by this medial structure. That such is not the case may be experimentally determined. The writer, in conjunction with Professor Elsberg, operated on a number of dogfish, detaching and completely removing the cerebellum mediale. The experiment animal was then replaced in the aquarium tank and its actions observed. The results were impressive, particularly because the animal did not manifest the amount of motor disturbance which had been anticipated from an operation causing the removal of the cerebellum. As a matter of fact, this experiment, repeated a number of times, showed clearly that the animal was well able to maintain its optimum physiologic position. It swam and rested belly down. Many of these animals lived for several days, and at no time did they manifest any tendency to lose the ability for maintaining this optimum posture. In every case, however, there was a peculiar change in the animal's

power of propulsion in swimming. It seemed to have lost the ability to coordinate the intersegmental body movements and progressed through the water wholly by means of its tail, very much as a boat is sculled by an oar from the stern. It differed entirely in its action from the swimming movements of the normal animal. The paired fins were used little, if any, and the rhythmical series of intersegmental body movements seemed to be entirely absent. If the animal was turned over on its back in the water, it immediately righted itself. If touched with a stick while resting, it would immediately swim forward, usually in a straight line, but in a more sluggish and uncertain manner than the normal animal when similarly treated. It showed no tendency to tip or turn while swimming, but proceeded with a manifest certainty maintaining its optimum physiologic position.

These experiments demonstrated that the cerebellum mediale was not connected with the motor apparatus necessary to maintain the posture best suited to the animal's purposes. They contrasted strikingly with the results obtained by Dr. Frederick Lee,⁷ who experimentally removed the semicircular canals and other portions of the vestibular apparatus in sharks. As a result the animals at once showed marked distortion in the function of maintaining the optimum physiologic posture. In swimming, depending on which canal was removed, they either turned around and around on their long axes, or tail over head, rotating about their transverse axes. This would seem to indicate that the semicircular canal apparatus is definitely related to the maintenance of the optimum physiologic posture. All of its connections are with the archeparencephalon (cerebellum bulbare and cerebellum jugale).

Our own experiments, revealing as they do a lack of all such disturbance, indicate that the new part of the cerebellum, cerebellum mediale, is active in some other function, which we have interpreted as the synergizing of intersegmental body movement with the lateral paired fins and the movements of the tail.

These complex activities essential to the extensive locomotor acts of the sharks and all true fish, represent a series of automatic associated movements which bring into action the metameric series of body segments, with the paired lateral fins and the tail. In these movements, as in all other purposive acts, the two essential streams of innervation may be detected: first, that which represents the purpose and design of the movement, and second, that which controls the posture pattern necessary to its execution.

7. Lee, F. S.: A Study of the Sense of Equilibrium in Fishes. Part 1. *J. Physiol.* **15**: No. 4, 1893; Part 2, *ibid.* **17**: Nos. 3 and 4, 1894. The Functions of the Ear and the Lateral Line in Fishes, *Am. J. Physiol.* **1**: No. 1, 1898. Ueber den gleichgesichtsinn, *Zentralbl. f. Physiol.*, Nov. 19, 1892.

In selachians, therefore (sharks and rays), a new part of the cerebellum over and above the archeparencephalon already described in cyclostomes, makes its appearance. This structure is intimately connected with some function other than that of maintaining the optimum physiologic posture. It is my present belief that the cerebellum mediale from the evolutionary standpoint represents what may be called the paleoparencephalon. It appears as the most conspicuous cerebellar element in all of the true fish, reptiles and birds. From the physiologic point of view, it is representative of the paleostatic element in the cerebellum, just as the cerebellum bulbare and cerebellum jugale represent the archestatic element in the most primitive vertebrate brains. This paleostatic function is concerned with the synergy of segmental (metameric) and segment (limbs, including fins, paddles and wings) musculature in the maintenance of concurrent postures necessary to inherent automatic associated movements. This paleostatic function is

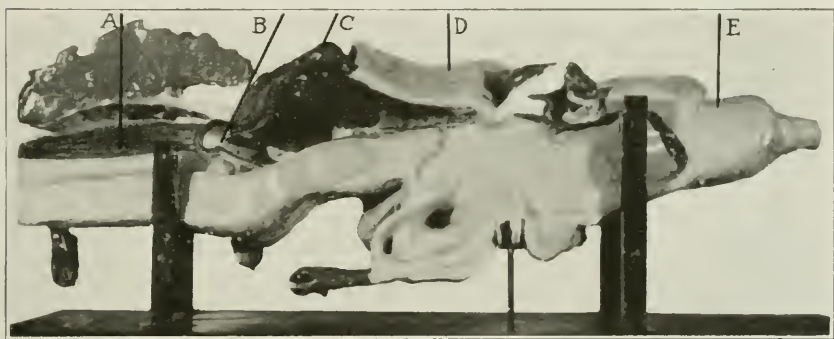


Fig. 8.—Reconstruction showing in midsagittal view the brain of a ganoid fish (*Amia calva*). This specimen illustrates the position of the archeparencephalon (cerebellum bulbare and cerebellum jugale) and the paleoparencephalon (cerebellum mediale). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, mesencephalon; *E*, telencephalon.

related to the paleokinetic system which, according to our present interpretation, has to do with the regulation of inherent automatic associated movements.

In Figure 8 is seen a reconstruction in midsagittal view of a brain in a ganoid fish (*Amia Calva*) in which the archeparencephalon and paleoparencephalon are shown. Figure 9 shows a dissection of the brain of a codfish, while Figure 10 represents a cross section of the brain of a codfish, showing the cerebellum bulbare, cerebellum jugale and cerebellum mediale.

An interesting sidelight on the progressive evolution of the cerebellum is afforded by the amphibia. The assumption of an amphibious

character of life with the partial establishment of a terrestrial habitat, appears in its inception to have concerned itself mostly with the adaptations to living on land. These adaptations were simple in the extreme and the motor capacities of the animal were limited, perhaps for lack of experience and opportunity, in their new environment. That the

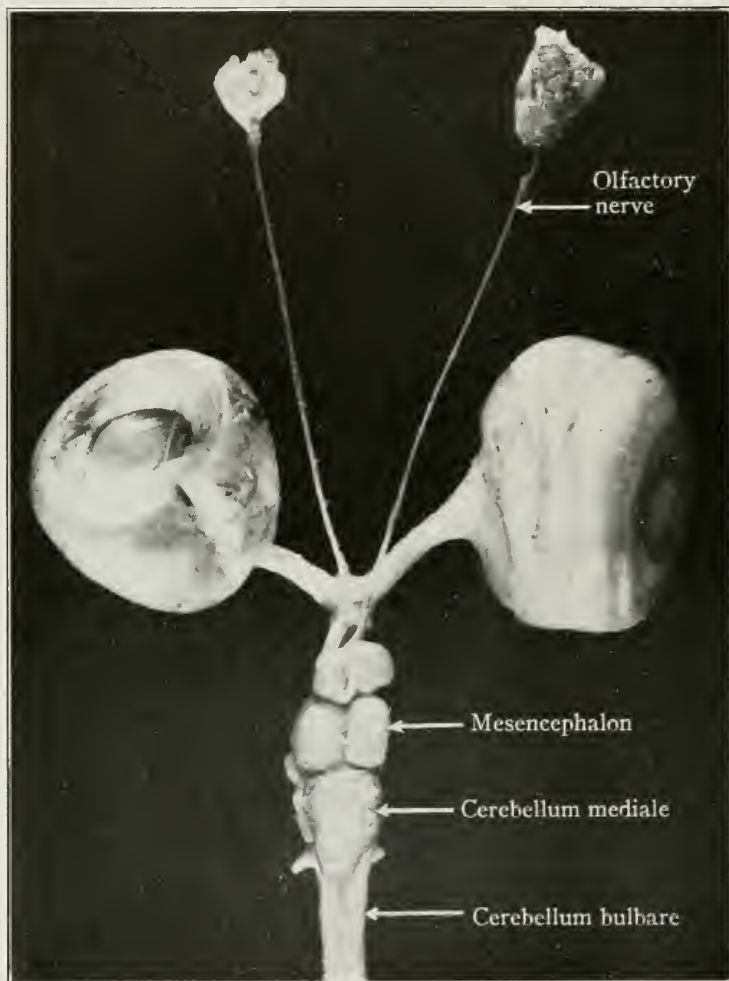


Fig. 9.—Dissection of cod fish brain with eyes and olfactory organs attached, showing cerebellum bulbare and cerebellum mediale.

transition from water-living to land-living was at once a decisive one probably does not express the fact. During the assumption of terrestrial life the process of transition was probably a slow one, taking the animal gradually out of the water to the soft marsh lands, until finally it was

able to maintain itself definitely on dry land. This intermediate period carried out in the marsh lands, provided an environment in which it was difficult to develop complex motor activities. It is for this reason that the amphibia in general, and particularly the anura (frogs and toads) have differentiated to such a slight degree in their cerebellar organization. They laid the foundations for life on dry land and were an extremely generalized type out of which terrestrial vertebrates took origin.

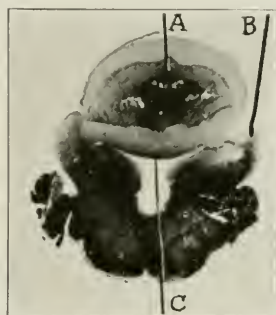


Fig. 10.—Cross section of the cerebellum in the cod fish. *A* indicates the cerebellum mediale; *B*, cerebellum bulbare; *C*, cerebellum jugale.

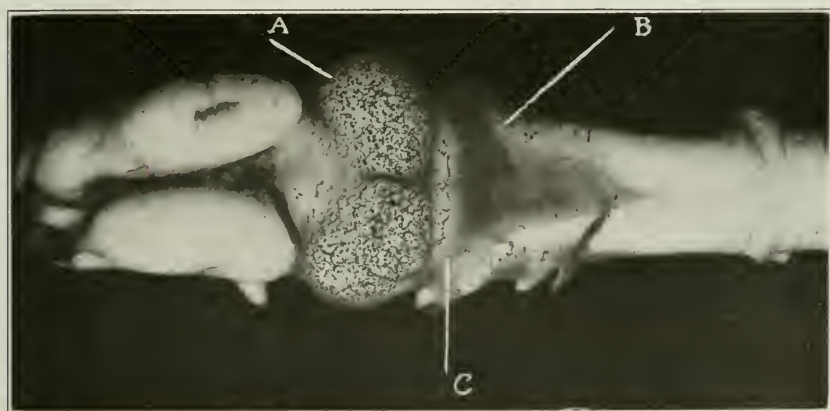


Fig. 11.—Dissection of frog's brain showing cerebellum bulbare and cerebellum mediale. *A* indicates mesencephalon; *B*, cerebellum bulbare; *C*, cerebellum mediale.

Figure 11 shows the cerebellum of the frog (*Rana sylvatica*). In cross section this cerebellum reveals the archeparencephalon (cerebellum bulbare and cerebellum jugale) with a small dorsal expansion representing the paleoparencephalon (cerebellum mediale). The simple

development of the cerebellum in amphibians has been a stumbling block in the explanation of the progressive evolution of the brain. When, however, the limited motor activities of amphibia in general are considered, it becomes clearer how their generalized cerebellar pattern was necessary as a stepping-stone for further advances in evolution.

In the advancement to the age of reptiles, little or no positive gain was made in increasing the capacity of locomotion. Whatever may



Fig. 12.—Dissection of alligator brain showing cerebellum bulbare and cerebellum mediale.

have been the character of the cerebellar organization in the gigantic tetrapod reptiles that inhabited the earth during the mesozoic age, it is certain that their modern congeners show little of progress in cerebellar evolution over the fish. The reptilian cerebellum, although it presents the three parts already observed in vertebrates lower in the scale, has expanded little as compared with the sharks.

Figure 12 shows a dissection of an alligator brain, and gives a good idea of the degree of development attained by reptiles generally speaking.

The next great step forward occurred in the birds, when a terrestrial habitat had become fully established. The development of the wings for flying and the necessary biped locomotion in consequence of this high specialization of the fore limbs introduced new problems in motion. In the main, the use of wings differs only in degree of complexity from the use of fins or paddles. The alar oscillations in flying are most delicately adjusted. The posture patterns of such complex automatic associated movements as those of flight require a corresponding complexity in the cerebellum mediale. This complexity is witnessed in the bird by the elaborate foliation of the cerebellum. In alighting, as well as in starting to fly, the bird has new problems of equilibrium. Its motor adjustments are even greater during locomotion on the ground. In the main, the actions of the hind limbs represent automatic associated movements of biped locomotion. On the other hand, most birds, and especially the ratite (running) birds, such as the ostrich and cassowary, have developed a high degree of independent action in each hind leg. They are able to use the claws in scratching for food and even to strike heavy blows with one leg while standing on the other. In certain birds, this same ability is witnessed in the wings, which are used in defense and attack. For the most part, however, the independent striking power of each wing is limited and probably belongs much more definitely to the type of automatic associated movement than to unilateral independent action.

It would seem, therefore, that in the birds the highest degree of unilateral independent action has been acquired in the legs, and this is probably reflected in a new element appearing for the first time in the avian cerebellum. A small lateral evagination from the cerebellum mediale, extending over the lateral aspect of the bulb on either side, marks the beginning of the cerebellum laterale. Many authorities have believed that this lateral extension of the cerebellum is the forerunner of the flocculus and is related to the movements of the tail. If such were the case, it might reasonably be expected that the cerebellum in all fish whose tail movements are complex should also have this lateral extension. The same objection would apply in forms like alligators and crocodiles; in fact, in all reptiles which use the tail extensively in locomotion. Comparative morphology does not lend support to the conception that this lateral extension of the avian cerebellum is concerned with the coordinative control of the tail. It is my opinion that these lateral expansions which appear in the later stages of embryonic development in birds as two lateral evaginations, represent entirely new elements in the cerebellar organization. They indicate the beginning of cerebellar control of unilateral independent movements in the legs.

The cerebellum of one of the running birds—the Australian ostrich—is shown in Figure 13. A cross-section of this brain in Figure 14 represents the compound nature of the organ, according to the lines already laid down. It illustrates the presence of the cerebellum bulbare, cerebellum jugale, cerebellum mediale, with the addition of this new part, the cerebellum laterale. The latter structure reaches

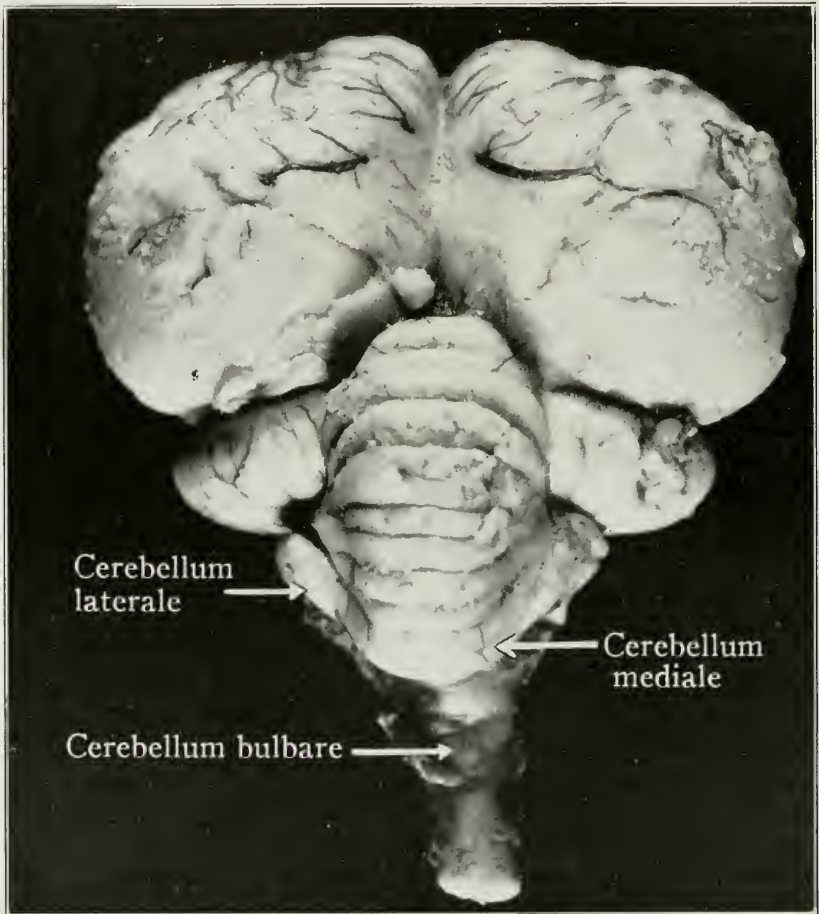


Fig. 13.—Dissection of emu brain (Australian ostrich) showing cerebellum bulbare, cerebellum mediale and cerebellum laterale.

its greatest development in the lateral lobes of the cerebellum in animals which have acquired the most highly differentiated fore limbs, whose distal extremities have developed as hands, and whose hind limbs serve the purposes of the most complete bipedal locomotion.

From the evolutionary standpoint, the cerebellum laterale represents the neoparencephalon. It rapidly expands in the different orders of mammals and reaches its highest development in the anthropoids and man. That the influences which underly its existence are directly connected with the differentiation of the fore and hind limbs, is shown by the progressive increase of nerve fibers entering the inferior cerebellar peduncle. Still further evidence is furnished by the development in the mammal of the middle cerebellar peduncle, which contributes its entire collection of fibers to the lateral cerebellar lobes. This peduncle is an index of the degree of connection between the cerebral cortex and the cortex of the cerebellum. It increases in volume in direct ratio to the

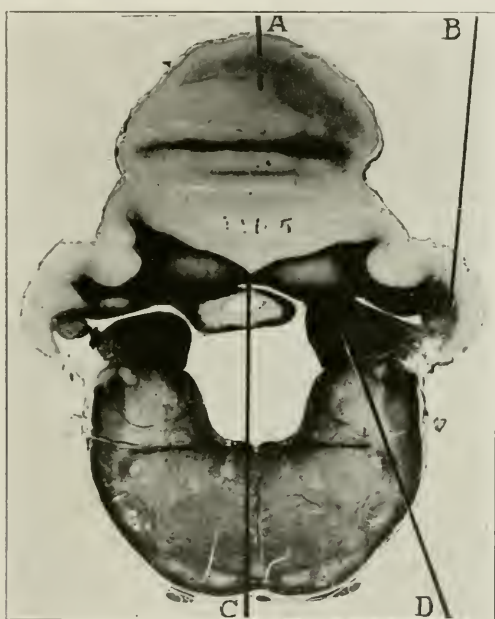


Fig. 14.—Cross section of the cerebellum in the emu (Australian ostrich). *A* indicates cerebellum mediale; *B*, cerebellum laterale; *C*, cerebellum jugale; *D*, cerebellum bulbare.

increase of the cerebral cortex, and attains its greatest dimensions in animals who have the most perfectly developed hands.

From the physiologic point of view, the neoparencephalon represents the neostatic cerebellar element. It is concerned with the synergy of the limb musculature necessary to the maintenance of the concurrent postures in acquired independent movements. The arms and legs in man and the anthropoids still retain many of the ancient automatic associated movements which are inherent. Their outstanding characteristic, however, is the fact that they are capable of developing many

complex movements which are acquired through constant repetition and concentration of attention. These skilled acts depend on the teachability of the cerebral cortex. They are highly individualistic rather than generic, and represent the ultimate independence of action in the limbs on the two sides of the body.

THE MAMMALIAN CEREBELLUM

A comparative study of the mammalian brain most convincingly reveals the relation of this capacity in skilled movement to the development of the cerebellum laterale. In the rabbit (Fig. 15) the lateral lobes are present but show a slight expansion only, in direct proportion to the degree of independent movement which this animal has acquired in the fore and hind limbs. The extremities are still used largely in the automatic associations of locomotion. This same observation applies

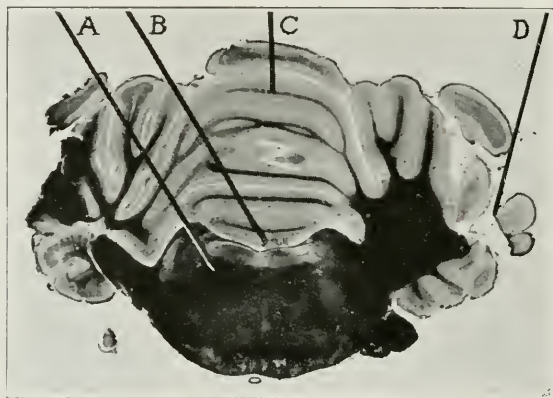


Fig. 15.—Cross section of the cerebellum in the rabbit. *A* indicates the cerebellum bulbare; *B*, the cerebellum jugale; *C*, the cerebellum mediale; *D*, the cerebellum laterale.

to all rodents, to ungulates and even to carnivores, although in the cat family there is a decided increase in the lateral cerebellar lobes in direct proportion as these animals have acquired more independent use of the fore limbs. In the transition from the carnivores to the primates, the lemur probably represents an intermediate stage. This animal shows an even more marked increase in its lateral cerebellar lobes than does the cat. It is still, however, a distinctly quadruped animal, and has freed its fore limbs but little for purposes other than locomotion. The cerebellum of the lemur is seen in Figure 16.

Very decisive is the advance observed in one of the new world monkeys (*Myecetes sineculus*, Fig. 17). This animal has acquired a considerable degree of independent skilled use of the hands.

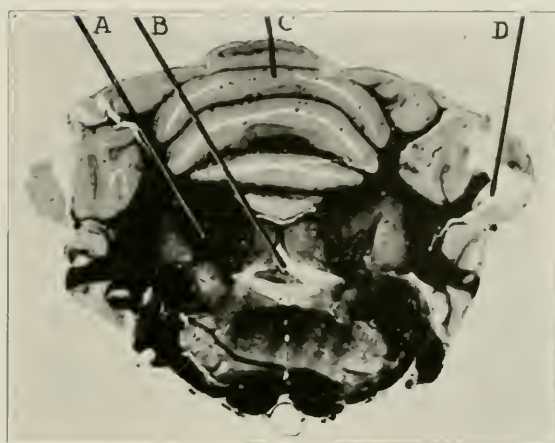


Fig. 16.—Cross section of the cerebellum in lemur. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

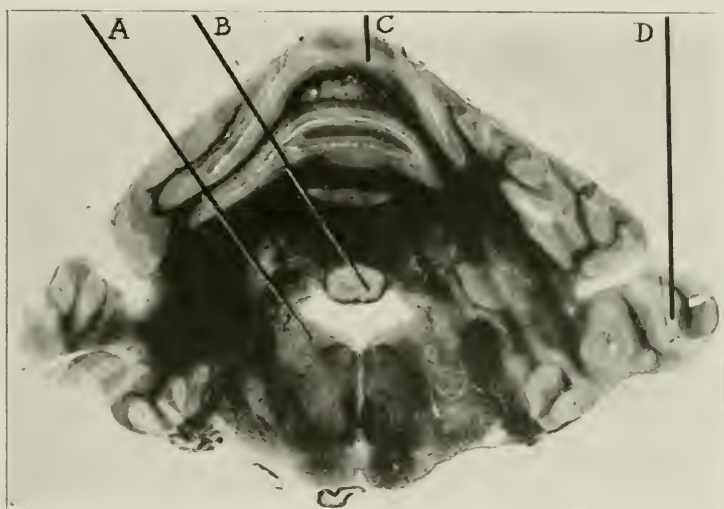


Fig. 17.—Cross section of the cerebellum in a South American monkey (*Myocetes sineculus*). *A* indicates cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

In the gibbon (Fig. 18), an early derivative of the common primate stock, the lateral lobes of the cerebellum have expanded still more. This animal is noted for its long arms and its well differentiated hands. These it uses not only for purposes of locomotion, but in many acquired skilled acts pertaining to defense and to the efforts of obtaining food.

In the three great anthropoids, orang-utan, chimpanzee and gorilla (Figs. 19, 20 and 21), the marked expansion of the lateral lobes of the cerebellum is clearly evident. The progressive approach to bipedal locomotion is well recognized in these animals, together with the freeing of the fore limbs for other purposes. How much the chimpanzee, and even the orang-utan, may be taught in the way of skilled movements with the arms and hands, is a matter of familiar knowledge. The

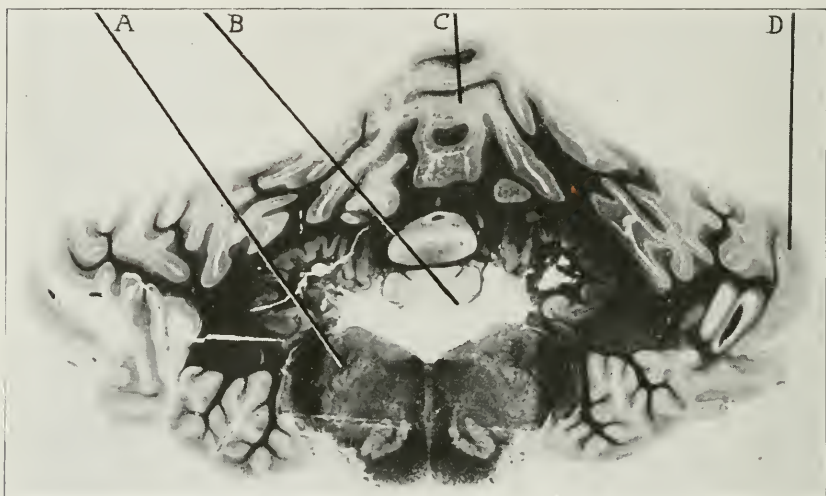


Fig. 18.—Cross section of the cerebellum in the gibbon. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

history of the gorilla, John Daniel, whose brain I am at present studying, affords an excellent example of the teachability of the anthropoid cortex. This animal was almost human in its manual achievements. The gorilla cerebellum here shown is that of Dinah, who died some time ago in the New York Zoological Gardens. This animal was only 3 years old, but had been taught to employ her hands with considerable skill, using a knife and fork, drinking from a cup and playing with a ball.

The final proof of the relation between the lateral lobes of the cerebellum and independent skilled movements, especially of the hands, is afforded by the human brain. A section of the cerebellar region in man is shown in Figure 22.

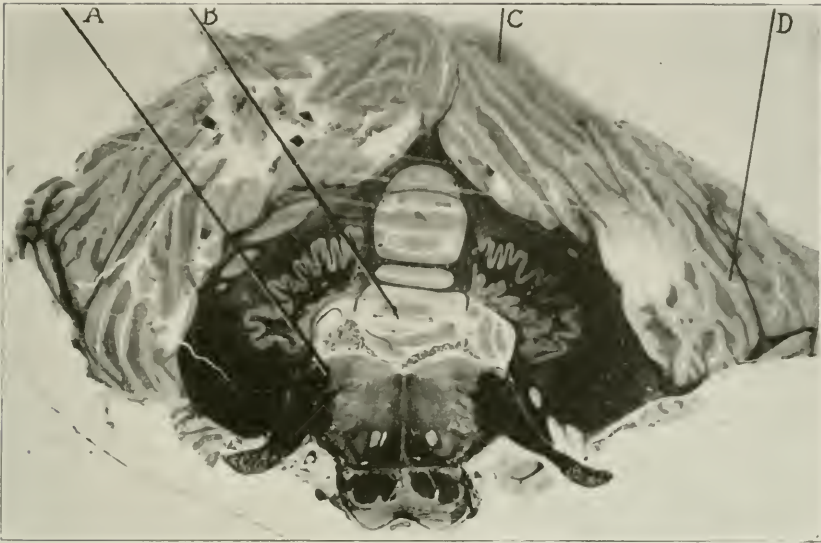


Fig. 19.—Cross section of the cerebellum in an orang-utan. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

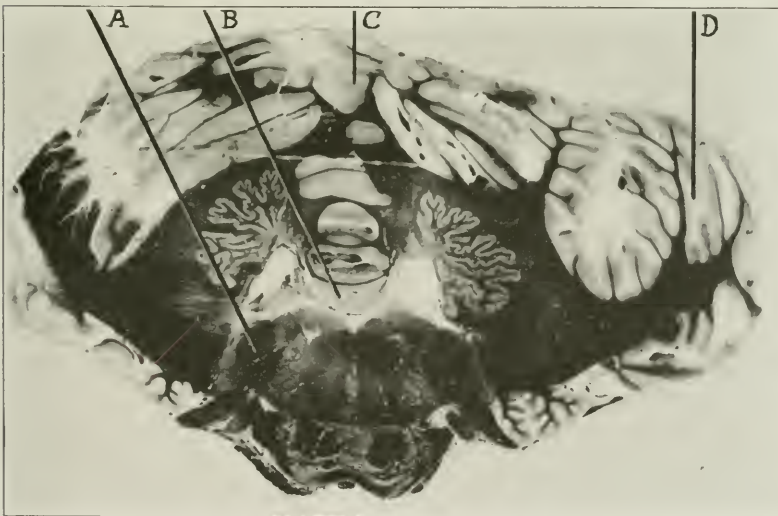


Fig. 20.—Cross section of the cerebellum in a chimpanzee. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

Such evidence as that afforded by the primates seems to substantiate the proposal that the neoparencephalon made its appearance in response to the development of the cerebral cortex. It seems to be directly related to the control necessary for skilled movements. Reviewing the conditions in the vertebrates, beginning with *Petromyzon* and continuing upward in the several orders of fish, it becomes apparent that the process of cerebellar expansion has passed through the evolutionary phase of the archeparencephalon to the epoch marked by the acquisition of the paleoparencephalon in selachians and all true fish. Then, with the appearance of birds, the lateral lobes had their inception and became more conspicuous in passing to the mammals. Through the mammalian orders, including monotremes, marsupials, rodents, carnivores and pri-

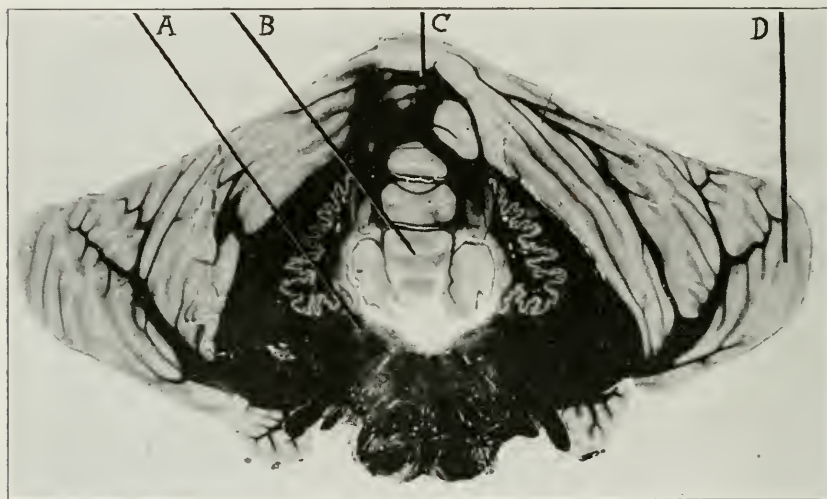


Fig. 21.—Cross section of the cerebellum in a gorilla. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

mates, the lateral lobes of the cerebellum have gradually expanded as the fore limbs have been progressively released from the functions of locomotion.

EXPERIMENTAL EVIDENCE

With the aim of controlling these tentative conclusions, several experiments were performed on animals, particularly with the idea of confirming the evolutionary interpretation of the cerebellum presented here. This work was done in collaboration with Professor Pike.

The first animal operated on was a cat in which the right semi-circular canals were destroyed. This operation aimed to interrupt the integrity of the apparatus connected with the archeparencephalon.

In consequence of the operation the animal was no longer able to maintain its optimum physiologic posture, although all of its movements in the effort to do so were well coordinated. It made vain attempts to get on its feet, to lie on its belly or to direct its head forward. These attempts resulted in constant but ineffective movements. This experiment illustrates what occurs when the archestatic system is disturbed.

In the second experiment, the lateral lobe of the cerebellum of a cat was destroyed on one side. Following the operation, the animal manifested an asynergy in the movements of the limbs on the same side as the operation. Although it was still able to maintain a proper physiologic posture, the movements of the hind and fore limbs on the

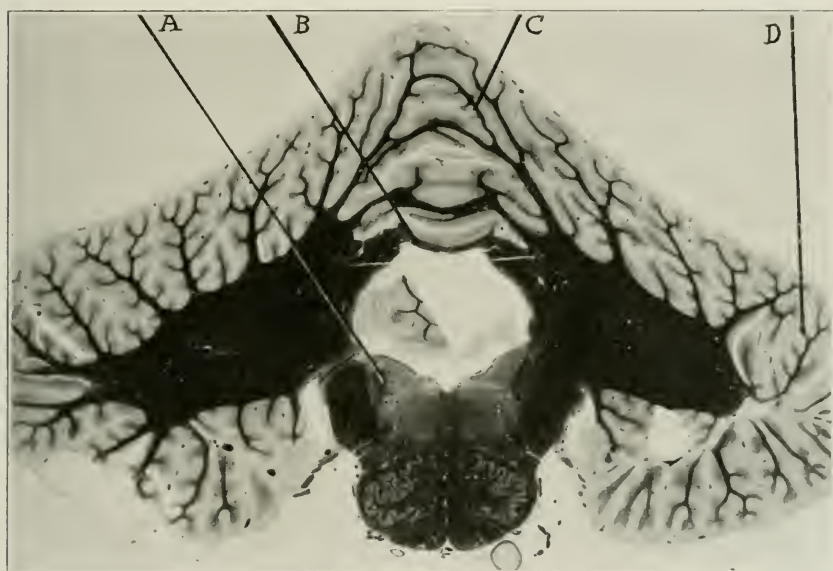


Fig. 22.—Cross section of the cerebellum in man. *A* indicates the cerebellum bulbare; *B*, cerebellum jugale; *C*, cerebellum mediale; *D*, cerebellum laterale.

same side as the lesion showed a marked dysmetria, because the stream of postures necessary to well coordinated movement no longer ran concurrently with the motor program of the animal's locomotor acts.

These two experiments bring into contrast the effects of disturbing, first, the archeparencephalon in its function of maintaining the optimum physiologic posture, and, second, the paleoparencephalon in its capacity of maintaining the postures necessary to the automatic associated movements of locomotion.

A second set of experiments with pigeons served to emphasize these conclusions. In one pigeon the semicircular canals on the right side

were removed. This animal maintained well coordinated movements, but was unable to hold itself in proper posture. In a second pigeon, the entire cerebellum, including all of its components, was removed. In this case not only was the power of maintaining the proper physiologic posture lost, but every movement with wings, legs, neck and tail showed a complete asynergy.

SUMMARY

It is my present belief that phylogenetically the cerebellum represents an integrative process of specialization. This process, as the result of increasing motor capacity during the progress of evolution, has centralized in one organ certain specialized structures acquired from time to time as need has been. The principal purpose of this mechanism has been the maintenance of posture in all the varied types of motor activity.

The cerebellar components which have developed during the several epochs of phylogeny and which have become more or less completely integrated within the ultimate cerebellum, are: (1) the cerebellum bulbare and the cerebellum jugale, representing the archeparencephalon; (2) the cerebellum mediale, representing the paleoparencephalon; and (3) the cerebellum laterale, representing the neoparencephalon.

The cerebellum bulbare and cerebellum jugale, the most primitive components of the cerebellum, took origin in the medulla oblongata. They are the sole representatives of cerebellar organization in the cyclostomes (lampreys and hagfish). Their homologues in the human brain are to be found in the vestibular nuclei of the medulla and the inferior cerebellar vermis. In man the greater portion of the cerebellum bulbare has become incorporated in the inferior vermis. Its bulbar remnants are seen in the vestibular nuclei.

The cerebellum mediale, the next component to appear, took origin from the roof of the medulla oblongata as a single unpaired evagination. It developed as the principal addition to the cerebellum bulbare and cerebellum jugale in all true fish, amphibians, reptiles and birds. Its homologue in the human brain is to be found in the superior cerebellar vermis.

The cerebellum laterale, the last component to appear, took origin in two lateral evaginations from the cerebellum mediale, symmetrically bilateral and one on either side. It forms an expansion of the cerebellum mediale and first appears in birds, but reaches its highest development in mammals, particularly in the primates. Its homologue in the human brain is to be found in the lateral lobes of the cerebellum.

The cerebellum bulbare and cerebellum jugale developed in the vertebrate as a central controlling structure of a mechanism necessary

to maintain the optimum physiologic posture of the animal. It became essential in consequence of the altered neuro-enteric relations determined by the invertebrovertebrate transition.

Having thus established a proclivity to centralize the control of posture-maintaining function, this general region of the brain acquired by evolutionary accession, the cerebellum mediale. This cerebellar component is essential to the posture-maintaining function of inherent automatic associated movements.

The cerebellum laterale developed to supply the necessary posture-maintaining function in the acquired, independent movements, especially of the arms and legs. It has reached its greatest development in man, whose capacity in acquired skilled acts with hands and feet far surpasses that of all other animals.

Each step in this evolutionary process has required the passage of age-long epochs of time. The following tabulation may make more graphic the stages which have been traversed in the phylogenetic achievement of this integrative process in the cerebellum.

ANALYSIS OF CEREBELLAR COMPONENTS

EVOLUTIONAL PHASE	MORPHOLOGIC ELEMENTS	PHYSIOLOGIC PHASE
Archeparencephalon (cyclostomes, lampreys and hagfish)	Cerebellum bulbare	Archeostatic: Synergy of dorsal, ventral and lateral musculature in maintenance of optimum physiologic posture; related to Archekinetic mechanism
From Silurian to Devonian	Cerebellum jugale	
Paleoparencephalon (all true fish, amphibia, reptiles and birds)	Cerebellum mediale	Paleostatic: Synergy of segmental and segment musculature (limbs) in maintenance of concurrent posture in inherent automatic associated movements; related to Paleokinetic mechanism
Through Devonian to Palaeocene		
Neoparencephalon (mammals)	Cerebellum laterale	Neostatic: Synergy of segment musculature (limbs) in maintenance of concurrent postures in acquired independent movements; related to Neokinetic mechanism
Palaeocene to recent		

A CLINICOPATHOLOGIC STUDY OF ACUTE AND CHRONIC CHOREA *

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Our main object in presenting this paper is to report the pathologic findings in two cases of chorea, one of the acute and one of the chronic variety. Until recent years the pathology of these two conditions has been elusive, and even today we do not have a clear conception of the underlying anatomic changes of the choreas; this is especially true of Sydenham's chorea.

Until Marie and Tretiakoff, in 1920, reported their case of Sydenham's chorea there was practically no known pathology in this condition so far as the nervous system was concerned. These writers described in their case findings identical with those seen in patients who died of epidemic encephalitis. In fact, as one reads the report of Marie and Tretiakoff one wonders whether their case was not one of the choreiform type of encephalitis.

One of us saw a case in the winter of 1920-1921 which he diagnosed as a severe case of Sydenham's chorea in a boy of 14, who later in the course of his disease developed lethargy and other typical signs of encephalitis from which he made a complete recovery. Had this patient died in the first stage of the infection, he would have been considered as having had Sydenham's chorea, and yet the outcome proved beyond a doubt that it was a case of epidemic encephalitis. As further evidence that Marie and Tretiakoff's case was not one of acute chorea may be mentioned the fact that there were no signs of endocarditis.

The possible relation between chorea and encephalitis has been discussed by Harvier and Levaditi, whose conclusions are: "1. Certain acute febrile choreas are brought about by the virus of encephalitis. 2. It is not yet proved that *all* acute febrile choreas are due to this agency."

REPORT OF CASES

CASE 1.—*History*.—A patient with Sydenham's chorea was admitted to the medical service of the hospital of the University of Pennsylvania on July 4, 1920, and was assigned to the service of Dr. Alfred Stengel.

On June 19, 1920, the patient, who was a girl, 12 years of age, began to have involuntary jerking of the upper extremities. Two or three days later

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the movements involved the head and the tongue and then the lower extremities. At the time of her admission to the hospital she could not talk and was confined to bed. Physical examination revealed a well nourished child with typical choreiform movements involving all four extremities, the trunk, the head and the tongue. A systolic murmur was heard at the mitral and pulmonic areas. The blood examination showed an eosinophilia of 6 per cent.



Fig. 1 (Case 2).—Horizontal section of hemispheres, showing dilatation of ventricle and atrophy of caudate and lenticular nuclei.

For six days following her admission her temperature was at or near normal, but after that it showed some variations, the temperature going as high as 101 to 102 F., and even as high as 105 degrees before death.

Necropsy Examination.—This revealed vegetations, semisoft but well attached on the auricular side of the mitral leaflets. These vegetations averaged 1 to 2 mm. in size. All the other valves were normal. A thorough and systematic

examination was made of the brain with special reference to the basal ganglions. In this study the toluidin blue, Weigert; phosphotungstic acid hematoxylin, Alzheimer-Mann, Marchi, Bielschowsky and hematoxylin and eosin stains were used. It is entirely proper to state at this point that absolutely no changes were found in the brain that could not be attributed to the acute febrile condition from which the patient suffered for some days before death. No changes were found which in any way approached the findings which Marie and Tretiakoff described in their case of Sydenham's chorea, a case which was probably one of epidemic encephalitis.



Fig. 2 (Case 2).—Basal ganglions. Note atrophic striatum, caudate nucleus and putamen and absence of internuclear fibers. *Nc.* indicates the caudate nucleus; *Put.*, the putamen; *Thal.*, the thalamus. Weigert stain.

A white man, 52 years of age, was admitted to the Philadelphia Hospital on Jan. 25, 1905, and died on March 3, 1917. His family history is one showing a marked degenerative defect as eight of his blood relations have had Huntington's chorea; other members of the family have been insane. The man's choreiform movements began in 1910, and shortly afterward he began to show mental symptoms, chiefly those of a simple dementia. The choreiform movements affected his entire body, and involved to a marked degree the muscles of his trunk and pelvis. His gait was rendered extremely bizarre by these movements, the unusual thing in his gait being a series of bowing movements. Some considered this a case of dystonia musculorum deformans.

Necropsy Examination.—The brain appeared to be what we have chosen to call "paretic looking." The cerebrum, cerebellum, brain stem and spinal cord were smaller than normal. The convolutions were atrophic and the fissures widened, this being especially evident over the frontorolandic areas. The pia-arachnoid was everywhere thickened, but to a more marked degree over the anterior half of the brain where it concealed the underlying structures by its opacity. Decortication could be performed without tearing the cortex.



Fig. 3 (Case 2).—Putamen, showing typical astrocytes, B. Phosphotungstic acid hematoxylin stain; $\times 368$.

Cross section of the brain in the usual manner showed that the ventricles were dilated (Fig. 1) and that the basal ganglia were small in proportion to the rest of the brain. The cortex was visibly narrowed, especially anteriorly.

The microscopic method of examination included large sections from different areas of the cerebrum, brain stem, cerebellum, spinal cord and serial sections through the basal ganglia and hypothalamic region. The stains used were the same as in the preceding case.

The Cortex.—Weigert stain showed atrophic convolutions separated by widened fissures and a narrow cortex with loss of many of the myelinated

fibers in all layers, most noticeable anteriorly. With toluidin blue the cyto-architecture was profoundly altered, so much so that the normal relationship could not be recognized. An occasional Betz cell remained to identify the motor area. The ganglion cells throughout, but especially in the fronto-rolandic cortex were decreased in number, and diseased. Those which remained exhibited various forms of acute and chronic cell change, such as simple chromatolysis, axonal degeneration, Nissl's severe cell disease and atrophy. The silver impregnation stains confirmed the findings obtained with toluidin blue. The vessels were not increased in number, but they showed distinct

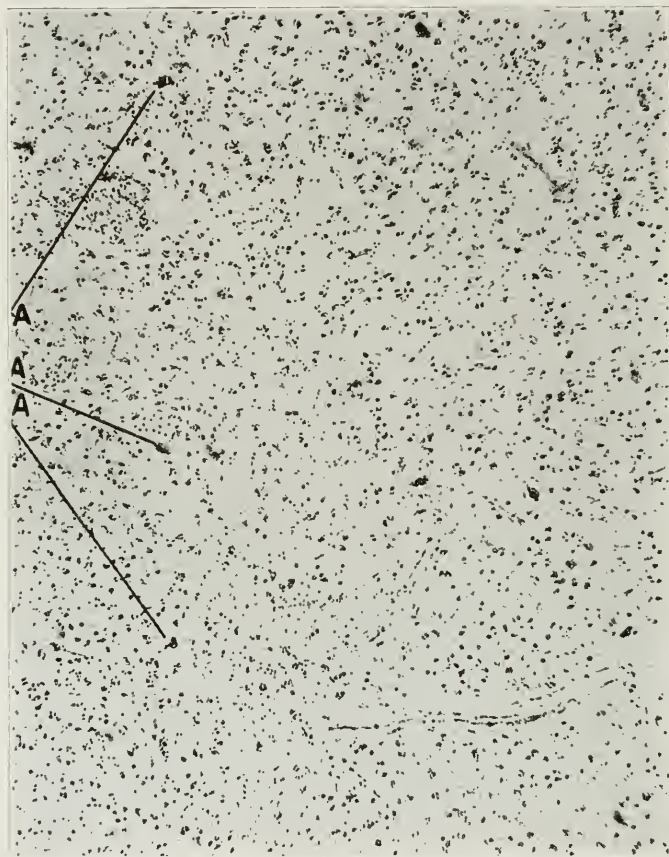


Fig. 4.—Normal putamen. Note satellitosis around large ganglion cells at *A*. Toluidin blue; $\times 57$. (Use hand lens.)

sclerosis and hyaline degeneration; for the most part the perivascular spaces were dilated, containing only the products of brain degeneration which at first glance simulated the cuffing with lymphocytes and plasma cells found in paresis. Glia cells were increased in number.

The pia-arachnoid showed distinct thickening, due to a connective tissue proliferation and containing within its meshes cells which on differential staining proved to be gitter cells with a tendency to agglutinate in the region of

fissures and about the vessels. Amyloid bodies were present, especially in the outer layers of the cortex and in relation to the ventricles, showing that there was a slow degenerative process going on. Although sought for, no foci of degeneration were found as described by Klebs, Oppenheim and Hoppe, Marie and Lhermitte and others; nor were plaques found such as are seen in senile dementia and at times in senility. The central white substance showed atrophy of the projection system of the cortex with replacement by neuroglia fibers arranged similarly to the destroyed fibers, showing that the process had taken place slowly.



Fig. 5 (Case 2).—Caudate nucleus. Note intense glial infiltration. Phosphotungstic acid hematoxylin stain; $\times 57$.

The Cerebellar System.—In the study of large sections of the cerebellum, including serial sections of the dentate nucleus, nothing of note was found except a smallness of all the structures. There were no areas of softening. The pontile nuclei, red nuclei and olives were intact.

The Central Ganglions.—As shown in Figure 2, with the Weigert stain there was marked atrophy of the central ganglions, particularly of the caudate and lenticular nuclei. As can be seen, the caudate was distinctly shrunken; the

putamen was apparently more involved than the globus pallidus. The thalamus was relatively intact. The internal capsule stained well, showing no areas of degeneration, and its dimensions were in proportion to the size of the brain. The internuclear fibers which take origin in the small cells of the caudate nucleus and putamen (the striatum of C. and O. Vogt) were absent. Serial sections through the hypothalamic region revealed no degeneration of the projection fibers (striothalamic, striosubthalamic and striomesencephalic radiations).

More minute histologic study showed that the nerve cells had practically all disappeared from the caudate and putamen, with only a few of the smaller and some of the larger motor type (Malone) of cells remaining. The phosphotungstic-acid hematoxylin stain showed an extreme proliferation of the glial elements, especially of the astrocytes (Fig. 3), which had a tendency to collect about the vessels. As could be seen in the gross, the globus pallidus (the pallidum of C. and O. Vogt) was much less involved. The characteristic large cells were present in the usual number. Glia cells were slightly increased, although astrocytes were rare. In the thalamus, there were atrophic lesions of the cells with a glial proliferation, but infinitely less intense than in the striatum. The vessels showed the same changes as found in the cortex—thickening of their walls, and occasional perivascular collections of degenerated material. No foci of degeneration were seen.

The spinal cord showed no pathologic condition, and the liver, except for congestion, was normal.

COMMENT

The changes found in the case of Huntington's chorea are distinct and rather widespread, involving the frontorolandic cortex, the meninges, the vessels, the caudate nucleus and putamen, with an escape of the globus pallidus and the optic thalamus. It is worth noting at this point that despite the extensive cortical and striatal changes, no secondary degeneration was found in the pyramidal tracts or in the extra-pyramidal system. The fact that the caudate and putamen are selected in this disease with an escape of the globus pallidus is easily explainable when it is realized that the striatum is developed from the same part and is really one body separated in man by the passage of the anterior fibers of the internal capsule. As is well known, the globus pallidus is composed of large ganglion cells, which Malone has shown to be related to the motor system in their general structure. Some of these cells are scattered in the caudate and putamen, and they are relatively uninvolved. It is the small type of cell which bears the brunt of the pathologic process.

We were disappointed in the absence of pathologic findings in the case of acute chorea, especially so because Marie and Tretiakoff found such marked alterations in their case. Our case was undoubtedly one of Sydenham's chorea, the diagnosis being based on the type of irregular movement seen, the presence of endocarditis and eosinophilia. The case was not one of encephalitis, because the lesions of that disease were absent. We believe that in the course of Sydenham's chorea cerebral changes must occur, but modern methods have so far failed to

reveal them. Care must be taken in the interpretation of the various sections taken from the basal ganglions, because in control sections made from relatively normal persons the same cell changes were demonstrated as in the case of Sydenham's chorea. As Bielschowsky has shown, the collection of cells around the large motor type of ganglion cell in the corpus striatum will occur under normal conditions, an indication of the fragility of these elements. This was found in the case of Huntington's chorea, but not to a greater extent than in a normal case. There was no appreciable change in the smaller type of cell in the case of Sydenham's chorea, in the specimen of which the subthalamic region was normal and no cortical alterations were noted. The vessels were all markedly congested, but not more than is usual in a patient dying of pneumonia or any other infectious disease. No thrombosed vessels were found.

The symptomatology of the corpus striatum is being slowly evolved, due in large measure to the work by S. A. K. Wilson, Mills, C. and O. Vogt, Hunt, Spiller, Marie and others. It is worth noting that our knowledge of the functions of the corpus striatum has been derived largely from clinicopathologic study, and while our presentation is of small moment, it may add a little to the sum total of our knowledge.

SUMMARY

1. A clinicopathologic study was made of a case of Sydenham's chorea and of one of Huntington's chorea.
2. In the typical case of Sydenham's chorea a mitral endocarditis was found. The pathology in the brain of acute cell changes with the marked congestion can be attributed to the acute infectious disease from which the patient died.
3. In the case of chronic chorea the process involved mainly the striatum (caudate and putamen) and the cortex; the changes being typical of a chronic degenerative process in these parts, selective in action.
4. No relationship could be found between the acute and chronic varieties of chorea.
5. The findings of epidemic encephalitis were not present in our case of Sydenham's chorea. When such are present, as in Marie's case, we believe them to be the result of the epidemic forms of encephalitis and not ordinary types of acute chorea.

TWO CASES OF BRAIN TUMOR WITH VENTRICULOGRAPHY

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CASE 1.—History.—A boy, 10 years old, was admitted to the Presbyterian Hospital on June 10, 1922, complaining of headache, vomiting, failing vision and numbness in the extremities. The headache had commenced a year previously, was chiefly frontal and was often accompanied by vomiting. Impairment of vision had been progressing steadily since early in May. Numbness of the limbs, especially on the right side, had been present for two weeks. Double vision had been complained of a year before and lasted several months.

Examination.—There was distinct bulging of the forehead, and the superficial veins of the forehead and temples were prominent. In this region there was also tenderness on precussion. Hearing was normal. There was no paralysis of the extremities, and coordination was normal. The tendon reflexes were absent in the legs and present in the arms. The abdominal and plantar reflexes were normal. Sensation was normal. Dr. T. D. Allen reported: Bilateral papillitis, swelling of right disk being 2.5 diopters; of the left disk, 2 diopters; vision on the right, 20/50; on the left, less than 1/200. The fields of the right eye were generally contracted from 10 to 30 degrees; the upper nasal field was entirely gone. In the left eye, there was only a small island remaining in the upper temporal field close to the point of fixation; central vision was lost. In the right eye, there was total paralysis of the sixth nerve; nearly total paralysis of the external branches of the third, and slight paralysis of the fourth. In the left eye, there was total paralysis of the fourth nerve; slight paralysis of the sixth, and nearly total paralysis of the external branches of the third nerve. Ptosis and nystagmus were not present. The spinal fluid was under greatly increased pressure—58 mm. of mercury—was clear and pale yellow. The cell count was 8. The Ross-Jones globulin test gave a faintly positive reaction. There was also a slight Lange reaction (0112221000). The Wassermann test was negative with both blood and spinal fluid. The blood showed slight leukocytosis (10,700 to 13,200). Roentgenologic examination of the head showed a definite outward bulging in the left temporal region, unusually prominent suture lines, and a suggestion of digital impressions.

As there was considerable uncertainty as to whether the suspected tumor was in the posterior fossa or in the frontal region, it was decided to resort to ventriculography. On June 16, a needle was inserted into the posterior horn of the right lateral ventricle through a trephine opening. Fifty c.c. of fluid were withdrawn and 40 c.c. of air injected. Roentgenograms were taken with the head on either side and one with the forehead down. No air was seen in the picture obtained in the last named position. In the lateral views the ventricles were found to be rather large, but one of the anterior horns contained no air, and the anterior border of this ventricle appeared uneven. This appearance was obtained when the left side of the head was against the plate, and theoretically the right anterior horn should have been the occluded one. However, the roentgenologists, by comparing the two lateral plates, concluded that the left anterior horn was the one affected, and that

the air had not been given time to rise to the opposite side. The procedure was well borne by the patient, and a week later Dr. Allen found some decrease in the swelling of the disks and a little improvement in the vision of the left eye. On June 26, Dr. Davis turned down a large flap over the left frontal lobe, but no growth was found. On inserting a needle, 50 c.c. of fluid were removed from the left anterior horn, which we had thought was occluded. For two days the patient was in very poor condition. Dr. Allen found a further reduction of swelling of the disks on the day after the operation, and on June 29, the swelling of the left disk was not over half a diopter. There was also a wider range of ocular movements. On June 27, the leukocyte count was 30,000 but the temperature was only 99.4 F. No fever developed, but the patient gradually failed, and he died on July 8.

Necropsy Examination.—At necropsy the brain was found to be under very high pressure. The meningeal vessels were practically empty, the gyri very



Fig. 1 (Case 1).—Symmetrical internal hydrocephalus.

flat, the sulci obliterated. A little pus was found in the left tympanum and mastoid.

After formaldehyd hardening and with empty ventricles the brain weighed 1,230 gm. On section, no change was found in the cerebrum except marked and perfectly symmetrical internal hydrocephalus. The enlargement of the third ventricle and aqueduct was especially marked (Fig. 1).

The under surface of both occipital lobes was raised like a dome and their gyri completely flattened by the upward pressure of the cerebellum. Externally the cerebellum showed no change except marked molding of the inferior portion at its line of contact with the foramen magnum.

On section, a large tumor mass was found to fill and greatly distend the upper part of the fourth ventricle (Fig. 2). The greatest dimensions of the tumor were 5 cm. from side to side and 3 cm. from above downward.

The lower part of the ventricle was not invaded. The upper portion of the tumor was dark reddish-black as a result of recent hemorrhage, while the lower portion was light gray. In its lower portion, the tumor appeared more firmly attached to the roof of the cavity than to its floor, while in the upper portion the opposite was true. The upper end of the tumor occupied the distended aqueduct of Sylvius and projected into the distended third ventricle.

Histologically, the tumor proved to be a glioma.

CASE 2.—*History*.—A married woman, 34 years old, was admitted to the hospital July 27, 1922, on the service of Dr. T. D. Allen, to whom we are indebted for the privilege of studying and reporting this case. Seven years before she began to have epileptic attacks, preceding which she saw flashes of light. The convulsions were general and were sometimes associated with biting of the tongue and evacuation of the bladder. In the beginning the attacks occurred every day, and often several times a day, but they gradually decreased in frequency and severity. There was a little headache after the

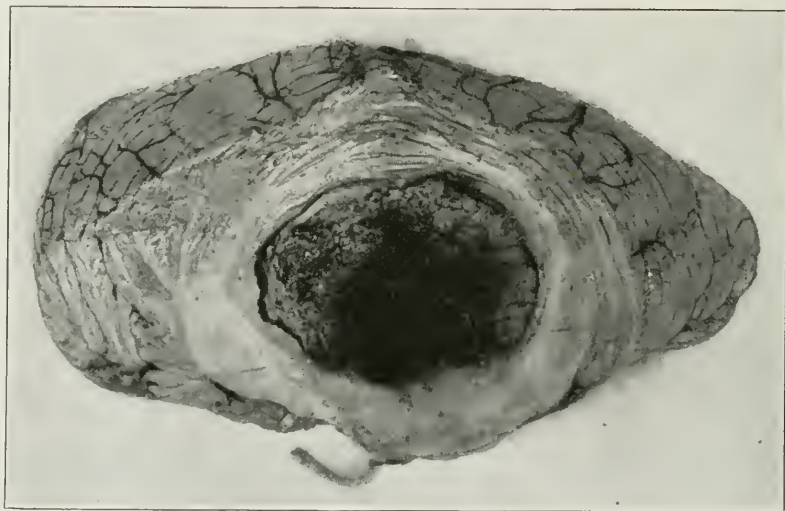


Fig. 2 (Case 1).—Tumor in upper part of fourth ventricle.

attacks, and sometimes at the beginning of the menstrual periods, but it never was severe, and in later years was infrequent. Vision began to fail about one year and seven months prior to admission, and during the last two months she had been practically blind, although some light perception was present part of the time. (As late as June 24, vision on the right was 20/100 and on the left 20/40.) This intermittence was very striking. At the time of admission, she began to complain of numbness and a pricking sensation in the right half of the face, including the mucous membranes of the nose, mouth and tongue. Formerly she had noticed a similar sensation in other parts of the body after the convulsions. Dr. Allen found that the pupils were small. The left pupil reacted normally, the right one somewhat sluggishly, to light. Bilateral papillitis was present with a swelling of 4 diopters of each disk. On examination, the tongue was found to protrude slightly to the left. The muscles of mastication were apparently weak on

both sides, but the patient did not complain of difficulty in chewing. There was no definite facial palsy, but the left eye was habitually wider open than the right. Hearing was good. Coordination was good. The tendon reflexes were practically normal; the left knee and ankle reflexes were slightly stronger than the right. The plantar reflexes were normal; the abdominal

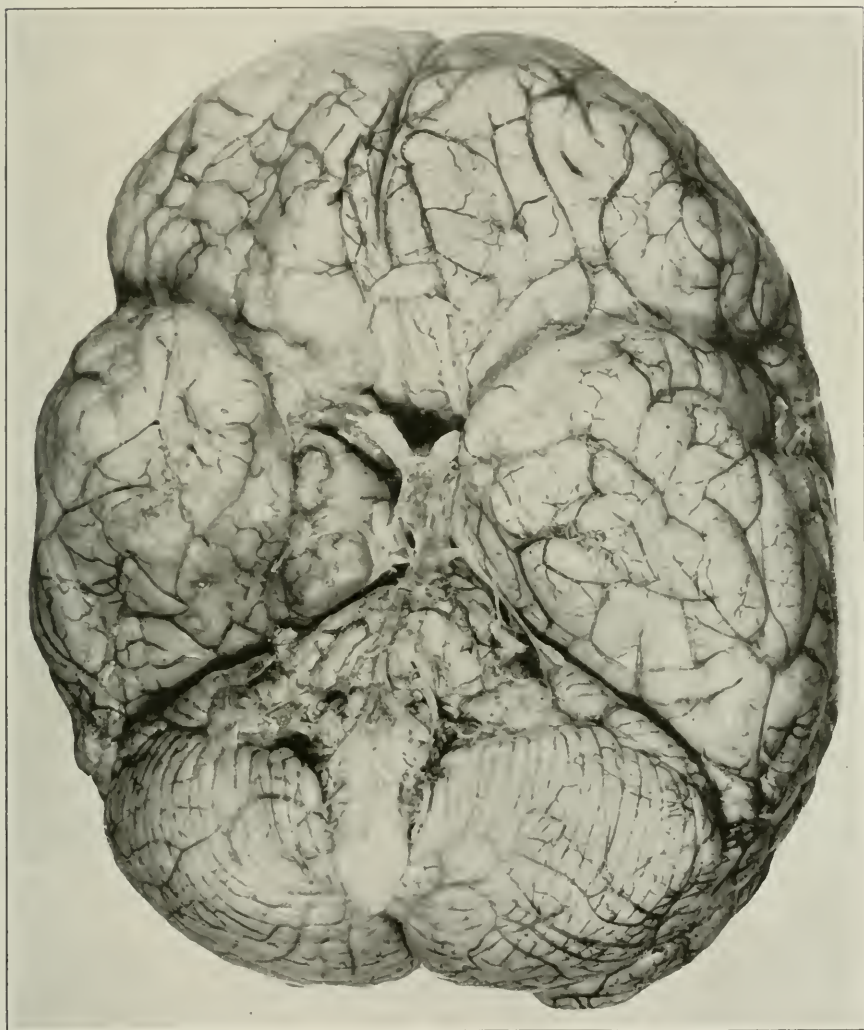


Fig. 3 (Case 2).—Base of brain showing tumor of right temporal and frontal lobes.

reflexes were diminished on the left. There was a questionable impairment of taste on the right half of the tongue and a very definite diminution of pain, touch and temperature sensation on the right side of the head, both on the face and posteriorly. There was less definite impairment of tactile and pain

sensation in all four extremities. The spinal fluid pressure was 12 mm. of mercury. All spinal fluid tests, as well as the blood Wassermann test, were negative. The leukocyte count was 13,400. On roentgenologic examination of the head no abnormality was noted, except that the posterior clinoid processes were indistinct. A tentative diagnosis was made of a tumor on the right side of the brain, so located as to make direct pressure on the chiasm, as it was not thought that the papillitis and blindness could be due to hydrocephalus or general increase in pressure. Although quite certain that the tumor was inaccessible, we yielded to the temptation of attempting to obtain a more definite localization by means of ventriculography. On August 15, Dr. Davis trephined and introduced air into the left lateral ventricle. Only about 20 c.c. of fluid was obtained, and possibly a slightly larger

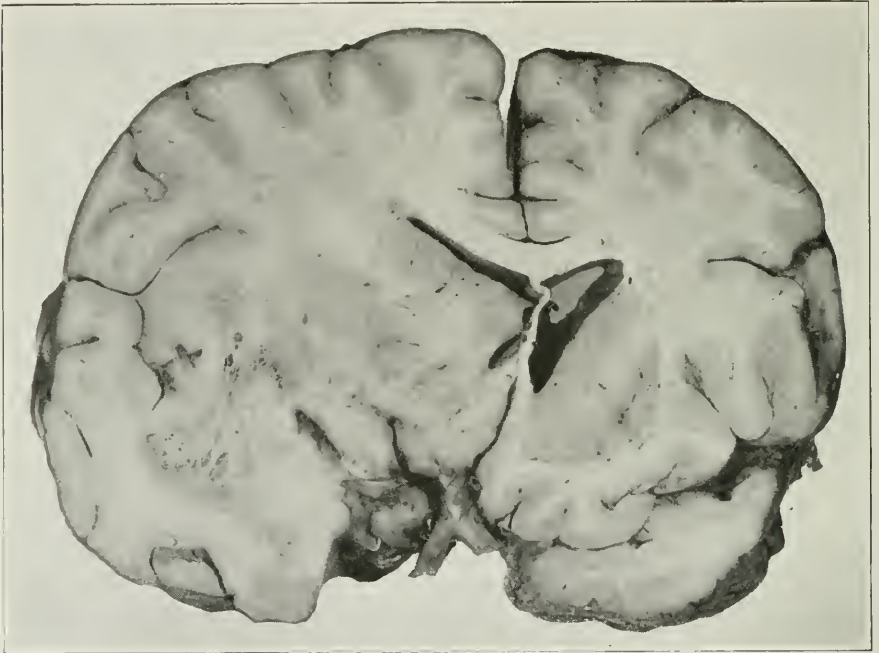


Fig. 4 (Case 2).—Cross section of tumor in temporal lobe.

amount of air was introduced. Films were taken with the head on either side, and with the forehead and occiput down against the plate. The description by the roentgenologist, Dr. Cassie B. Rose, omitting reference to the trephine holes, is as follows: "Film against the right side of the skull seems to show good filling of the lateral ventricle. Lateral view, with the left side against the film also shows the lateral ventricle except that the anterior horn is not made out. On anteroposterior view, there is on the left side a long, narrow shadow, probably a filling in the left ventricle, with a streak extending toward the right side. The right ventricle, however, is not made out. On postero-anterior view a decreased density is seen above the left orbit, probably the shadow of air in the left ventricle; right not made out."

On returning from the operating room, the patient, was somewhat cyanotic, but the pulse was regular and full. One hour later the pulse rate suddenly rose from 72 to 145, and the cyanosis increased. Twenty minutes later the patient died.

Necropsy Examination.—The dura was everywhere tense. The superior longitudinal sinus was empty and the brain squeezed tightly against the dura. The gyri were flat and the sulci largely obliterated.

The hardened brain weighed 1,475 gm. The right hemisphere appeared to be larger than the left, especially in the anterior portion. Looking at the inferior surface, we noted a grayish, rounded projection, firmer and slightly darker than the normal cortex, pressing against the anterior portion of the pons and displacing the chiasm toward the left (Fig. 3).

The fifth cranial nerve at its exit from the pons was in close contact with this growth. The right third nerve lay in a groove on the under surface of the projecting mass. The under surface of the right frontal lobe from a distance of 2 cm. in front of the chiasm also appeared darker than the surrounding brain tissue. The length of the whole tumor area along the posterior surface of the brain was 7 cm., its greatest width was 2 cm.

On section, the right temporal and frontal lobes were found to be occupied by an infiltrating, indistinctly-outlined, in places softened, tumor mass (Fig. 4). It invaded the cortex of the inferior surfaces and occupied roughly the lower two thirds of the right frontal lobe and the mesial two thirds of the temporal lobe. The ventricles on the right side were small, and, with the exception of the posterior horn and the end of the descending horn, were nearly obliterated. The lateral ventricle on the left side was not distended, and its widest point was seen at the cross-section illustrated in Figure 4.

In the occipital lobe, a few small hemorrhages marked the path of the ventricular puncture.

The cerebellum showed no gross change, except considerable molding at its contact with the foramen magnum.

Histologically, the tumor proved to be a glioma.

COMMENT

We report these cases chiefly to point out that in the hands of novices like ourselves ventriculography is likely to be misleading and dangerous. In the first case, sufficient air had not been introduced into the posterior horn to fill the anterior horns completely. In the second case, in which the ventricles were unexpectedly small, we evidently introduced too much air, and death was caused by the acute compression. Aside from determining the absence of hydrocephalus, we did not learn to locate the tumor any more accurately than we had by the neurologic symptoms. However, we are willing to admit that in selected cases and in hands more skilful than ours the method occasionally will lead to a correct localization and successful removal of a tumor which cannot be located by other known methods.

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THE DIFFERENCE BETWEEN A MUSCULAR AND A NEUROMUSCULAR INTERPRETATION OF WALKING*

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TABLE OF CONTENTS

- I. INTRODUCTION.
- II. ANATOMIC AND PHYSIOLOGIC BASES OF THE NEUROMUSCULAR MECHANISM.
- III. TECHNIC USED IN ANALYZING THE PROCESS OF WALKING.
- IV. MOVEMENTS DURING WALKING.
 - Finlike movement at hip.
 - Flexion reflex element.
 - Support element.
 - Movements of knee.
 - Flexion of knee in position of support.
 - Identity of homolateral movements of arm and leg.
- V. SUMMARY.

I. INTRODUCTION

Walking is a highly complicated process. The underlying mechanisms, in view of their importance in all studies of motility, have attracted attention at least since the days of the Weber brothers, nearly one hundred years ago. The general conception of the movements of the four extremities has not changed essentially since it was first described. These movements were recently described thus: "The right upper and left lower limbs are coupled together and swing forwards or backwards at the same time, flexion of one arm occurring with extension of the leg on the same side."¹ In other words, both the homolateral limbs as well as the pairs of arms and legs, respectively, move in opposite directions. This conception is based on ordinary observations and on the action of certain definite muscles. Though it has been recognized since the days of Huxley that the front of the leg is, in reality, the dorsal surface and though this has been abundantly proved by various anatomists who have studied the comparative anatomy and embryology of the limbs, the significance of this has apparently attracted little attention among neurologists and has not been used in the interpretation of group movements, normal or abnormal, of man. The process of walking has therefore been discussed from the point of view of muscular action, on one hand, and

* Read by title at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

1. Riddoch, George, and Buzzard, E. Farquhar: Reflex Movements and Postural Reactions in Quadriplegia and Hemiplegia, with Especial Reference to Those of the Upper Limb, *Brain* **44**:462, 1921.

from the point of view of its general control as a reflex mechanism by the spinal cord on the other. The classical work of Sherrington has established definite laws for this reflex action and has also indicated which muscles are involved in certain phases of reflex stepping (Fig. 1, Table 1). However, in all this, no attempt seems to have been made to interpret walking from a point of view which considers the embryology and evolutionary origin of the muscles involved. I shall attempt to show that the spinal cord controls, by its integrations, large groups of muscles which are related, not only embryologically, but also by virtue of their common nerve supply. On this basis it may be shown that the integrations controlling walking are among the foundations of the nervous system and constitute primitive motility patterns. All

TABLE 1.—THE MUSCLES SHOWN IN FIGURE 1 ARRANGED IN VENTRAL AND DORSAL GROUPS *

A		B	
Ventral	Dorsal	Ventral	Dorsal
I.	I. Psoas M. Gluteus minimus R. Rectus femoris F. Tensor fasciae femoris brevis S. Sartorius lateralis S'. Sartorius medialis	0. Quadratus femoris 3. Adductor minor 4. Adductor major (a part) 5. Semimembranosus 6. Biceps femoris pos- terior	
II. B. Biceps femoris posterior G. Gracilis T. Semitendinosus			1. Crureus 2. Vasti
III.	A. Tibialis anticus L. Extensor longus digi- torum P. Peroneus longus E. Extensor digitorum brevis	7. Gastrocnemius 8. Soleus 9. Flexor longus digi- torum	

* The first group in both tables includes muscles acting at the hip, the second those acting at the knee, the third those acting at the ankle.

the neurons engaged in producing patterns necessary to gait are of spinal origin. On these are superimposed other patterns appearing later in the course of development and more complicated mechanically.

In order to appreciate the significance of the various integrations controlling motility, it is necessary first to understand the most primitive of these and the manner in which they control muscles. Emerging from this method of interpretation of movement—which has been designated the neuromuscular ² and which considers the grouping of the peripheral motor neurons and their end-organs, the muscles—the idea

2. Kraus, Walter M.: A Principle Hitherto Undescribed of the Physiology of Movement and Posture. The Primitive Integration of Movement in Vertebrates, Arch. Neurol. & Psychiat. 7:381 (March) 1922.

will be developed that though the actual visible movements involved in walking indicate that in reality the homolateral limbs move in opposite directions, when their control by the nervous system is considered, these opposite movements are equivalent to movement in the same direction. Furthermore, it will be shown that there exist corresponding opposite phases of gait in which the analogues of the activity of singly hinged appendages in primitive animals can be made out, and

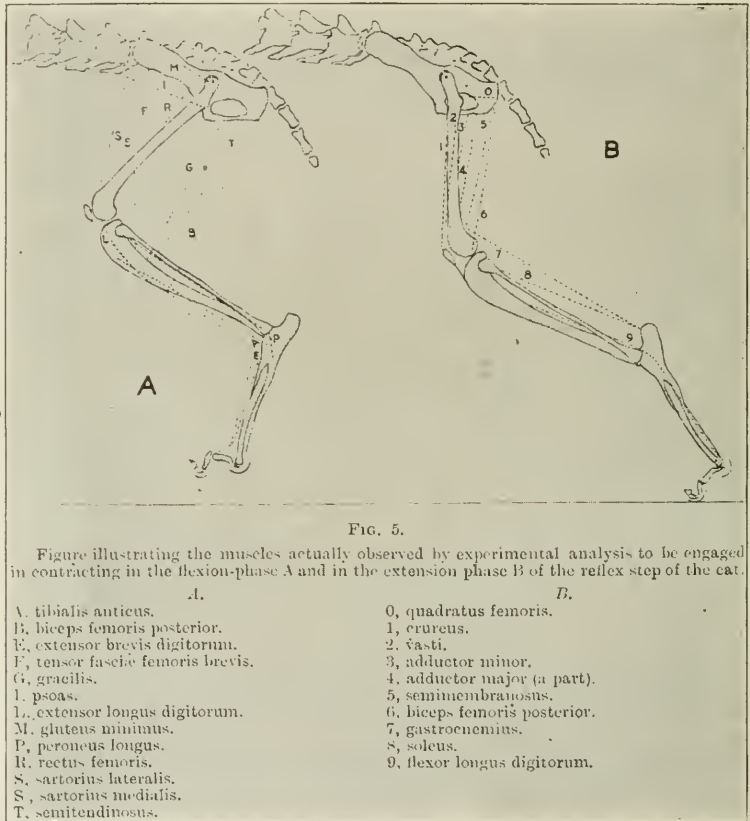


FIG. 5.

Figure illustrating the muscles actually observed by experimental analysis to be engaged in contracting in the flexion-phase A and in the extension phase B of the reflex step of the cat.

Fig. 1.—Reproduced from *Brain*, volume 33, page 22, 1910, from the article by C. S. Sherrington on "Remarks on the Reflex Mechanisms of the Step."

that superimposed on these is another pair of corresponding opposites also controlled by the spinal cord and based on the development, in the course of evolution, of the three-hinged appendages of the land-living vertebrate. Such an analysis of the control of walking by spinal integrations demands an entirely new point of view toward posture and progression patterns, not only in normal man but also in all disorders of the central nervous system in which these neurons are disturbed.

II. THE ANATOMIC AND PHYSIOLOGIC BASES OF THE NEUROMUSCULAR MECHANISM

As has been said before,² movements are now described in such terms as extension, abduction, internal rotation, etc. In other words, we describe the activity of a neuromuscular mechanism in terms of the muscular part of that mechanism alone, disregarding the neurologic element of movement and its control by the spinal cord and its peripheral nerves. The neural part of the mechanism is represented by peripheral motor neurons. The activation of groups of these neurons in various ways by the spinal cord is carried out by neurons within the spinal cord and is known as integration. When we describe in terms of muscular action only, complicated movements guided by integrations of the nervous system at levels limited to or higher than the spinal cord,

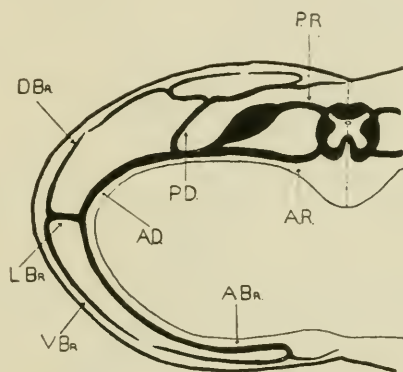


Fig. 2.—Adapted from "Human Anatomy" by G. A. Piersol, Part 2, p. 1284, Fig. 1085. *D. Br.*, dorsal branch; *V. Br.*, ventral branch; *L. Br.*, lateral branch; *A. Br.*, anterior branch; *P. R.*, posterior root; *A. R.*, anterior root; *P. D.*, posterior division; *A. D.*, anterior division.

we omit consideration of the spinal patterns and control of the final neuromuscular mechanism.

The nervous system is unquestionably built up of a series of physiologic levels of which the higher or more recently developed, phylogenetically speaking, carry out functions which are different from the lower. As Herbert Spencer remarked, "Integration keeps pace with differentiation." As animals become more and more differentiated, more and more needful of performing new kinds of movements in pattern, integrations appear to carry out these group movements. It is my opinion that we must have some conception of the integrating action of the lowest levels in order to interpret the higher. We must know how the spinal cord integrates movements in order to understand the activities and patterns of higher integrations superimposed in the course of

evolution and ultimately to understand why it has become necessary for these higher integrations to appear at all. It is necessary to consider in detail the general plan of the peripheral nerves, the general plan of the innervation of muscles by them and the embryologic grouping of the muscles themselves in order properly to examine integration of movement by the spinal cord.

A typical thoracic spinal nerve having nothing to do with the innervation of limbs shows a number of divisions after union of the anterior and posterior spinal roots. The trunk so formed divides into an anterior and posterior division. The anterior division divides further into a lateral and an anterior branch, the lateral branch dividing into a dorsal

TABLE 2.—DIVISION OF THE NERVES OF THE EXTREMITIES INTO DORSAL AND VENTRAL GROUPS

Origin	Upper Extremity	
	Groups	Nerves
Brachial plexus	Dorsal trunks (posterior cord)	Dorsal scapular Long thoracic Suprascapular Subscapular (2) Thoracodorsal Axillary Radial
	Ventral trunks (lateral and medial cords)	Nerve to subclavius Anterior thoracic (2) Musculocutaneous Median Ulnar
Lumbosacral plexus	Lower Extremity	
	Groups	Nerves
	Dorsal trunks	Superior gluteal Inferior gluteal Nerve to piriformis Femoral Peroneal
	Ventral trunks	Obturator Nerve to obturator internus and superior gemellus Nerve to quadratus femoris and inferior gemellus Tibial

and a ventral (Fig. 2). As far as the axial musculature is concerned, the portions of it developed from the ventrolateral portions of the embryo are supplied by the anterior division and its branches, while the dorsal musculature is supplied by the posterior branches. Thus, a simple scheme results from a physiologic point of view. There emerges the notion that, in general, movements of extension are guided by the posterior division, while flexion is guided by the anterior division. The lateral and rotatory movements produced by these groups of muscles are due to overactivity of the right and left halves of either the dorsal or the ventral groups or both. One theory assumes that the limb plexuses are made up only of the lateral branch of this division. For the purposes of this discussion, it is not important to try to reach a decision as to which of these theories is correct.

The primitive extremities of the embryo are flattened buds having a dorsal and ventral surface. The "premuscle mass" on the dorsal side gives rise to muscles the majority of which, in the adult, have an extensor function. The "premuscle mass" on the ventral side gives rise to muscles the majority of which have a flexor function. The

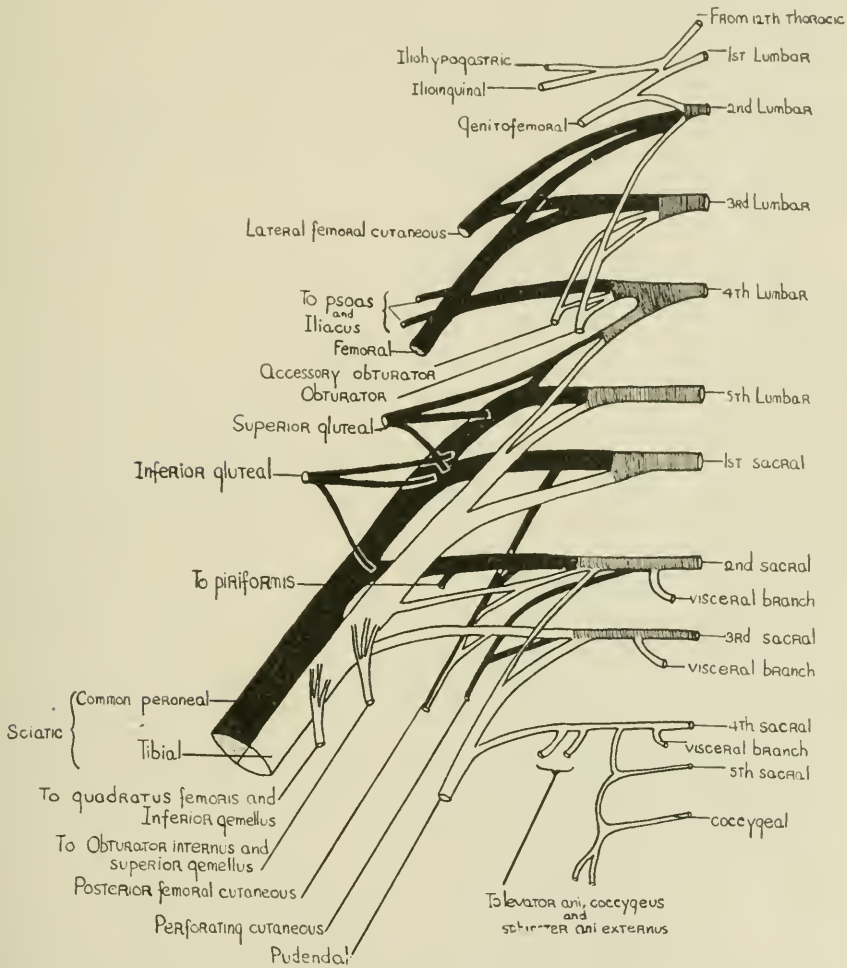


Fig 3.—Plan of the lumbar, sacral and pudendal plexuses. Black, dorsal; white, ventral; cross-hatched, combined. (Modified from Gray's "Anatomy," pp. 949 and 958).

crux of the whole situation lies in the fact that certain muscles have a function opposite to that which would be expected from a knowledge of the side of the limb bud (ventral or dorsal) from which they develop. For example, the iliopsoas group develops from the dorsal

muscle mass of the lower limb bud, but has a ventral function, that is, flexion.² This is an old and well-known fact, and is due to rotation of the leg in the course of evolution.

To return now to the question of innervation, we find that the embryology of the muscles is beautifully emphasized by their innervation. It is well known that the nerves of the brachial and lumbosacral plexuses divide into two definite groups, one of which is posterior or dorsal and the other anterior or ventral. In the arm, the ventral branches of the brachial plexus innervate the subclavius and pectoral muscles as well as all the muscles supplied by the musculocutaneous, median and ulnar nerves. The remaining nerves are dorsal (Table 2). The same division in the lumbosacral plexus is indicated by Figure 3 and Table 2.

A detailed table of muscles based on their derivation from the dorsal or ventral aspects of the limb bud and their supply by the dorsal or ventral branches of the limb plexuses shows that those muscles which develop from the dorsal layers of the bud are supplied by the dorsal branches of the limb plexus and those which develop from the ventral layers by the ventral branches. The analogy to the condition in the axial musculature is obvious (Tables 2, 3, 4 and 5).³

From all this we arrive at some possibility of schematization. In the nerves and muscles of the upper and lower limbs we find, as in the case of the iliopsoas mentioned before, that muscles which would be expected to have an extensor action have in reality a flexor action and vice versa. In other words, were we to group the muscles acting in any given movement merely on the basis of their activity as muscles and leave out of consideration their embryology and activating nerves, we would be leaving out entirely the division of these nerves and of their end-organs, the muscles, into large ventral and dorsal groups.

To illustrate how this information may be used for discovering just how the spinal cord may integrate movement, let us consider the use of relatively simple spinal integrations in the process of walking.

III. TECHNIC USED IN ANALYZING THE PROCESS OF WALKING

Simple observation of walking and running in a normal man does not permit of a precise analysis of the various phases of gait. The movements are too rapid and the transitions between flexion and extension, etc., are too small to give accurate results. Advantage was there-

3. Lewandowsky, M.: *Handbuch der Neurologie*. Flatau, Edward: *Die Motorische, Sensible und Reflex Segmentierung im Rückenmark*. *Allg. Neurol.* **1**, Pt. 2, p. 659, 1910. Cunningham, D. J.: *Textbook of Anatomy*, New York, William Wood & Co., 1918, p. 743. Braus, Hermann: *Anatomie des Menschen, Bewegungsapparat*, Berlin **1**: 1921.

fore taken of cinematographic pictures by which a considerable number of successive phases of walking and running could be obtained.

The pictures taken were a series from *X* to *Z* (Fig. 4). *X* *Y* and *Z* were indicated on a black drop by sheets of white paper. *Y* indicates

TABLE 3.—DIVISION OF THE MUSCLES OF THE UPPER EXTREMITY INTO DORSAL AND VENTRAL GROUPS

Dorsal		Ventral	
1	Levator scapulae	1	
2	Serratus anterior	2	
3	Rhomboides major	3	
4	Rhomboides minor	4	
5	Supraspinatus	5	
6	Infraspinatus	6	
7	Teres minor	7	
8	Deltoid	8	
9	Subscapularis	9	
10	Teres major	10	
11	Latissimus dorsi	11	
12		12	Subclavius
13		13	Pectoralis major
14		14	Pectoralis minor
15		15	Biceps brachii
16		16	Brachialis *
17		17	Coracobrachialis
18	Brachioradialis	18	
19	Extensor carpi radialis longus	19	
20	Extensor carpi radialis brevis	20	
21	Supinator	21	
22	Extensor pollicis longus	22	
23	Extensor indicis proprius	23	
24	Abductor pollicis longus	24	
25	Extensor pollicis brevis	25	
26	Extensor communis digitorum	26	
27	Extensor carpi ulnaris	27	
28	Extensor minimi digiti quinti	28	
29	Anconeus	29	
30	Triceps	30	
31		31	Pronator teres
32		32	Flexor carpi radialis
33		33	Palmaris longus
34		34	Flexor digitorum sublimis
35		35	Flexor digitorum profundus
36		36	Flexor pollicis longus
37		37	Pronator quadratus
38		38	Lumbricalis 1
39		39	Lumbricalis 2
40		40	Lumbricalis 3
41		41	Lumbricalis 4
42		42	Abductor pollicis brevis
43		43	Opponens pollicis
44		44	Flexor pollicis brevis (lateral head)
45		45	Flexor pollicis brevis (medial head)
46		46	Flexor carpi ulnaris
47		47	Adductor pollicis obliquus
48		48	Adductor pollicis transversus
49		49	Interosseus volaris 1
50		50	Interosseus volaris 2
51		51	Interosseus volaris 3
52		52	Interosseus dorsalis 1
53		53	Interosseus dorsalis 2
54		54	Interosseus dorsalis 3
55		55	Interosseus dorsalis 4
56		56	Opponens digiti quinti
57		57	Flexor digiti quinti brevis
58		58	Abductor digiti quinti

* Supplied by both dorsal and ventral nerves.

the point at which the subject was directly in front of the camera and *X* and *Z* two points equidistant from *Y* on either side. The distance *X*—*Z* is equal to 17 feet. In order to include positions *X* and *Z*, a panoramic series was used. Of the series reproduced, seventeen pictures were

TABLE 4.—DIVISION OF THE MUSCLES OF THE LOWER EXTREMITY INTO DORSAL AND VENTRAL GROUPS

Dorsal		Ventral	
1	Iliacus	1	
2	Psoas major	2	
3	Psoas minor	3	
4	Pectineus *	4	
5	Sartorius	5	
6	Rectus femoris	6	
7	Vastus lateralis	7	
8	Vastus medialis	8	
9	Vastus intermedius	9	
10	Tensor fasciae latae	10	
11	Gluteus minimus	11	
12	Gluteus medius	12	
13	Piriformis	13	
14	Gluteus maximus	14	
15	Biceps femoris, short head	15	
16		16	Biceps femoris, long head
17		17	Semitendinosus
18		18	Semimembranosus
19		19	Adductor magnus
20		20	Obturator externus
21		21	Adductor longus
22		22	Adductor brevis
23		23	Gracilis
24		24	Obturator internus
25		25	Superior gemellus
26		26	Inferior gemellus
27		27	Quadratus femoris
28	Tibialis anterior	28	
29	Extensor hallucis longus	29	
30	Extensor digitorum longus	30	
31	Peroneus tertius	31	
32	Peroneus longus	32	
33	Peroneus brevis	33	
34	Extensor digitorum brevis	34	
35		35	Gastrocnemius
36		36	Soleus
37		37	Plantaris
38		38	Popliteus
39		39	Tibialis posterior
40		40	Flexor digitorum longus
41		41	Flexor hallucis longus
42		42	Quadratus plantae
43		43	Abductor digiti quinti
44		44	Flexor digiti quinti brevis
45		45	Opponens digiti quinti
46		46	Plantar interosseus 1
47		47	Plantar interosseus 2
48		48	Plantar interosseus 3
49		49	Dorsal interosseus 1
50		50	Dorsal interosseus 2
51		51	Dorsal interosseus 3
52		52	Dorsal interosseus 4
53		53	Adductor hallucis
54		54	Lumbricalis 1
55		55	Lumbricalis 2
56		56	Lumbricalis 3
57		57	Lumbricalis 4
58		58	Abductor hallucis
59		59	Flexor digitorum brevis
60		60	Flexor hallucis brevis

* Supplied by both dorsal and ventral nerves.

used for analysis. It required three and one-fifth seconds to walk from X to Z and about one second to walk one pace. By a stroke of good luck the picture showing one leg in support and the other in maximum flexion (No. 25) was but one from Y, that is, almost directly in front of the camera (Figs. 4 and 5). The degree of rotation of the body appearing at X and Z is shown in Figure 6. This may be compared to Figure 5.

TABLE 5.—DIVISION OF THE MUSCLES OF THE AXIS INTO DORSAL AND VENTRAL GROUPS

	Ventral	Dorsal
Muscles of the Back:		
Serratus posterior superior.....		+
Serratus posterior inferior.....		+
Splenius		+
Sacrospinalis		+
Iliocostalis		+
Longissimus		+
Spinalis dorsi		+
Semispinalis		+
Multifidus		+
Obliquus capitis inferior.....		+
Obliquus capitis superior.....		+
Rectus capitis posterior major.....		+
Rectus capitis posterior minor.....		+
Rotatores		+
Interspinales		+
Intertransversarii	+	
Trapezius		+
Muscles of the Neck:		
Scalenus anterior	+	
Scalenus medius	+	
Scalenus posterior	+	
Longus capitis	+	
Rectus capitis anterior	+	
Longus colli	+	
Rectus capitis lateralis	+	
Sterno-kleido-mastoid	+	
Muscles of the Thorax:		
Intercostales	+	
Levatores costarum	+	
Subcostales	+	
Transversus thoracis	+	
Muscles of the Abdominal Wall:		
Obliquus externus abdominis.....	±	
Obliquus internus abdominis.....	+	
Cremaster	+	
Transversus abdominis	+	
Pyramidalis abdominis	+	
Rectus abdominis	+	
Quadratus lumborum	+	
Muscles of the Perineum:		
Sphincter ani externus.....	+	
Corrugator cutis ani.....	+	
Transversus perinei superficialis.....	+	
Bulbocavernosus	+	
Ischiocavernosus	+	
Sphincter urethrae membranacea.....	+	
Transversus perinei profundus.....	+	
Muscles of the Pelvis:		
Levator ani	+	
Coccygeus	+	

Except for careful mathematical analysis, this rotation is not important, though in all analyses it should be regarded as a source of error in drawing conclusions. Before taking the pictures it was determined that the difference between having the subject walk part of the circumference of a circle indicated by X, W, Z (Fig. 4), and walking a straight line, X, Y, Z, was so slight as to make its carrying out unimportant. This difference, the distance from Y to W, is only a foot and a half.

A series of cinematographic pictures was projected one after another on a wall at a distance of 12 feet. The images produced were 19 inches high. The method of determining the successive movements or changes in the angular relations of the thigh and torso, or of the knee and the thigh, was to trace a series of pictures with, in the first instance, the torso of each successive picture superimposed on the preceding one. The thigh movements are thus made out (Figs. 7 and 8). In tracing the thigh-knee relations, the thigh was taken as a constant

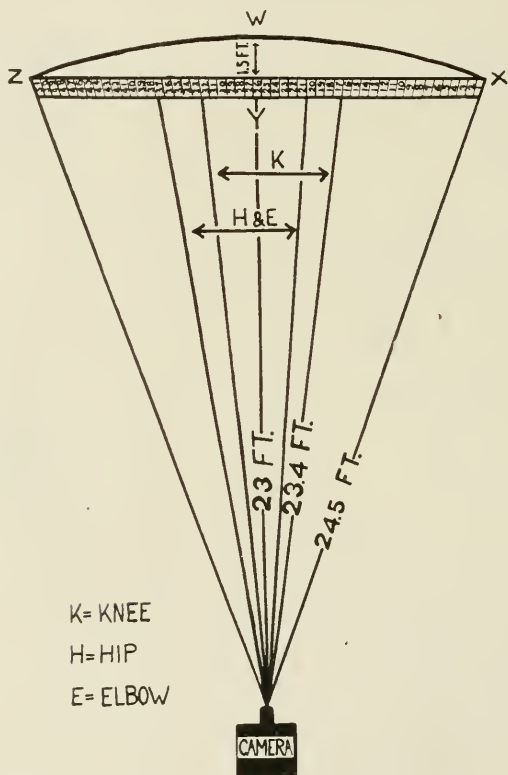


Fig. 4.—Diagram representing the distance relations of the patient to the camera. The patient walked a straight line from X to Z. The small numbers between X and Z each represent a picture. For further explanation see text, section III.

(Figs. 9 and 10). The elbow movements were determined by the same method (Fig. 11). Because of rotation, the shoulder movements cannot be illustrated easily and have been omitted. Certain precautions must be taken in such successive tracings in which changes in the positions of muscles cause changes in contour. The difference in the lower posterior thigh line, caused by changing positions of the hamstrings,

is illustrated in Figure 12, in that *A* shows the contour in flexion at the knee, *B* the contour in extension at the knee. In all drawings the anterior thigh line was used as the standard.

The horizontal has been inserted in all the pictures of the torso-thigh series. In the thigh-knee series the horizontal for the first tracing of each series has been inserted. A vertical line through the central position between ventral and dorsal and at right angles to the floor line was put into the torso-thigh cuts to indicate tilting of the torso and the relative change of the center of gravity in these positions.

It is obvious that a complete pace must be taken for analysis. It so happened that seventeen pictures were needed to show this. If,

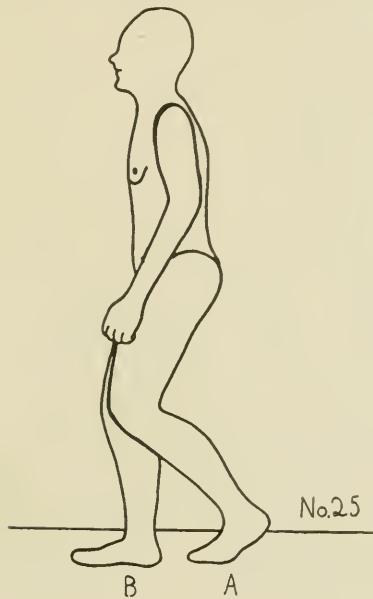


Fig. 5.—This tracing shows the left leg, *A*, in the maximum position of the flexion reflex and the right leg, *B*, in the maximum position of support. These constitute the third and fourth phases of walking. The actual picture, as well as the positions, are mathematically midway between those shown in Figures 13 and 14. The thighs are seen to be in alinement with one another. Flexion at the knee of the right leg, *B*, is apparent.

therefore, the first and seventeenth are the same, the ninth is the middle picture and must represent a stage midway between, and one in which the legs are in mirrored positions of those in the first and seventeenth. Furthermore, it would be expected that midway between the first and ninth and the ninth and seventeenth there would occur the maximum position of flexion reflex in one leg and a maximum position of support in the other. The analysis bore this out, and the fifth and thirteenth

represented these. To check this, they were compared. The arm positions were also used to determine the accuracy of the conclusions. When the arms are parallel to one another, one leg is in maximum support, *B*, and the other in maximum flexion, *A* (Fig. 5). An analysis of succeeding pictures furnishes a check to the selection of the various stages of the pace, irrespective of any suppositions on purely mathematical grounds. Still another method of determining by analysis of the knee movements that the fifth and thirteenth are indeed maximum positions of flexion reflex and support will be illustrated below. The actual pictures used were numbers 21 to 37 for the hip, shoulder and elbow and numbers 17 to 33 for the knee. A slightly

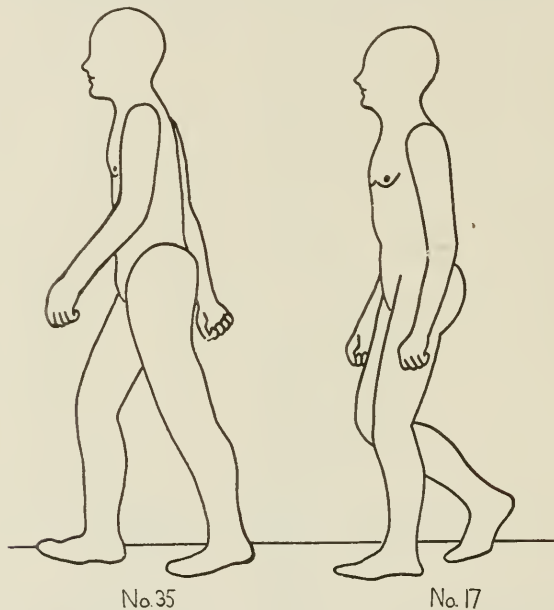


Fig. 6.—Rotation produced in a panoramic series of pictures, due to the patient's walking in a straight line. To avoid this rotation the patient should walk along the arc of a circle (Fig. 4, *X*, *II'*, *Z*).

different series was taken for the knee in order to emphasize the difference in the extent of oscillation during the posture and flexion phases.

IV. MOVEMENTS DURING WALKING

For the purposes of the present analysis of the activity of the legs in walking, the assumption has been made that it is composed of four simple activities, namely: (1) the ventral, finlike movement of the lower limbs at the hip; (2) the dorsal, finlike movement of the lower limbs at the hip; (3) the flexion reflex element, and (4) the support element.

Finlike Movement at the Hip.—For the moment let us omit the last two elements. If the assumption concerning these four activities be true, one may expect that in the process of walking the leg undergoes a pendulum-like action from the points of attachment of the leg at the transverse plane of the hip. Such an action was postulated in 1836 by the brothers Weber, though they attributed this activity not to muscular but to gravity effects. Both Vierordt and Duchenne of Boulogne have considered that this activity is a part of the process of walking, carried out not by gravity, but by muscles. In an analysis of a complete pace of a normal person by means of tracings of successive cinematographic pictures, it has been found that in the process of walking the movement at the hip is continuous (Figs. 7 and 8).

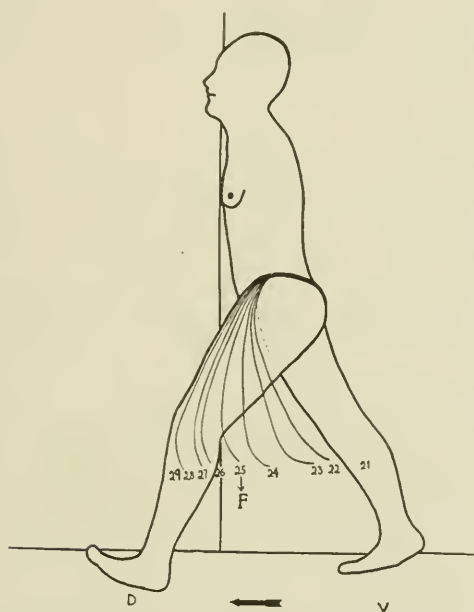


Fig. 7.—Consecutive tracings of the movement of the thigh in its progress from the ventral to the dorsal position. Compare with Figure 8.

It is well recognized now that the singly-hinged appendages of the fish preceded the three-jointed appendages of land vertebrates, such as amphibia. The most primitive grouping of movements by the spinal cord in the process of progression must have been one which correlated the activities of muscles much simpler than our own which lay on the ventral and dorsal aspects of the appendages, respectively. It seems obvious that the two groups of muscles which carried the fins in opposite directions must be controlled by spinal neurons permitting reciprocal innervation, so that when an agonist contracts its antagonist

is relatively relaxed. In order to realize this primitive finlike movement of a singly hinged appendage in man, it is necessary to regard the human leg as a rod and to ascertain whether there is a stage in the process of walking at which it assumes either a forward (dorsal) or backward (ventral) position. It is also necessary to determine whether these positions occur at regular intervals and whether they appear in definite relation to the two other elements of gait described by Sherrington as the extensor and flexor phases (Fig. 1).

It has been found that these two ventral and dorsal positions of either leg regarded as a rod do occur at regular intervals in the course of walking (Figs. 13 and 14), and it is found, furthermore, that

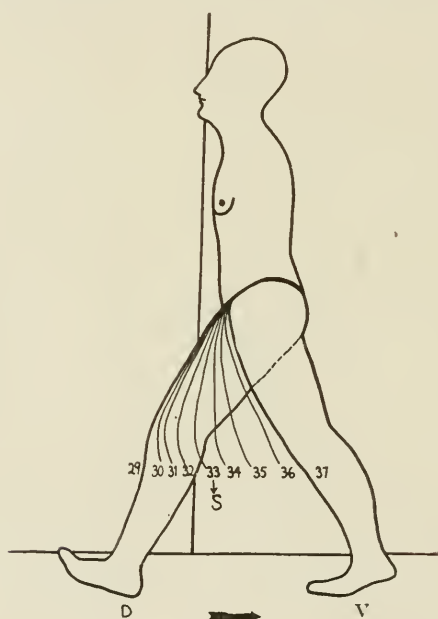


Fig. 8.—Consecutive tracings of the movement of the thigh in its progress from the dorsal to the ventral position. Compare with Figure 7.

midway between the appearance of the ventral and dorsal positions the flexion reflex element occurs, while the support element occurs midway between the appearance of the dorsal and ventral positions. It seems apparent, therefore, that in the process of walking the two primitive finlike movements at the hips can be made out with ease. That they occur rhythmically, that they are diphasic, continuous and represent alternating movements of the dorsal and ventral groups of hip muscles is illustrated by Figures 7 and 8. The rodlike positions are seen only at two stages of gait and are concealed at all other stages, due to the addition of the support and flexion elements.

The Flexion Reflex Element.—Since these movements which have just been described are necessarily more primitive than those of the three-jointed limbs of the land animal, it is assumed that the elements in walking developed by the land animal are considerably more complex and do not represent simple pendulum-like activities. As a matter of fact, the flexion reflex element represents an alternation of movement at the three great joints. This is beautifully demonstrated by an examination of the muscles given by Sherrington⁴ in his paper, "Remarks on the Reflex Mechanism of the Step," as those causing the extensor and flexor phases of gait. What Sherrington called the flexion phase of reflex stepping corresponds precisely to the third element of gait in this analysis. Figure 1 and Table 1, taken from Sherrington's paper, illustrate this. The table of muscles, to which there has been added the words "ventral" and "dorsal," indicates the alternation of movement—dorsal at the hip, ventral at the knee and dorsal at the ankle.

The Support Element.—The extensor phase which Sherrington described shows precisely the opposite picture. That omission of the neuromuscular mechanism may lead to an attempt physiologically to group movements obviously not susceptible to such grouping is by now apparent. Two examples of such an attempt appear in Walshe's⁵ paper on "The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs." He says:

I have said above that all the flexors of the limb are activated in the flexion reflex. These include hip and knee flexors and the dorsiflexors of the ankle and digits. Now, of these, the dorsiflexors, in anatomical nomenclature, are named extensors. There can be no doubt, however, that these muscles must be grouped physiologically with the flexors. . . . In view of the fact that the "extensor response" is a part of a general movement of flexion while the toe movement in the normal plantar response, the "flexor response," is, physiologically considered, a movement of extension, it is unfortunate that the accepted terminology is the direct converse of the facts. The expressions "extensor response" and "flexor response" are widely used by neurologists in this country, and I have therefore been compelled to make use of them, though it is very difficult to prevent confusion in the mind of the reader when speaking of a "crossed extension reflex" and of a "crossed extensor response"; two very different phenomena involving antagonistic groups of muscles. I wish, therefore, to emphasize that these empirical and misleading names are used here in their accepted sense, and have no physiological connotation such as the terms "flexion reflex" and "crossed extension reflex" have. I have consequently placed them in inverted commas to avoid as far as possible all confusion.

4. Sherrington, C. S.: Remarks on the Reflex Mechanism of the Step, *Brain* **33**:22, 1910-1911.

5. Walshe, F. M. R.: The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, *Brain* **37**:269, 1914-1915.

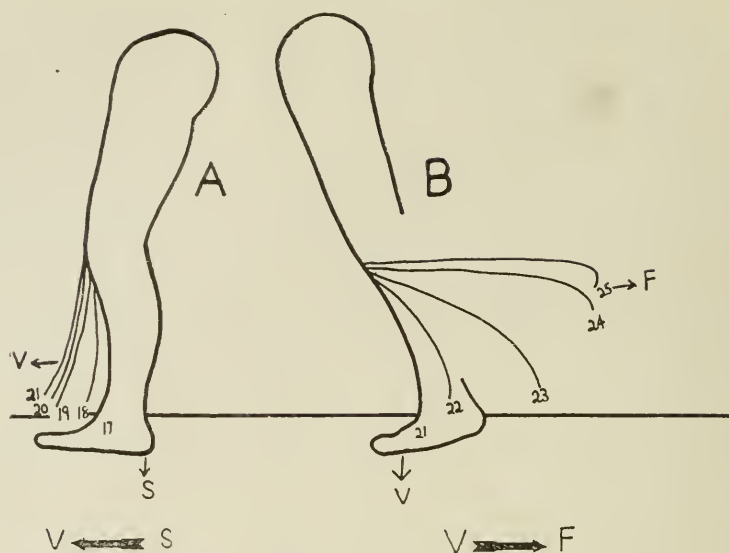


Figure 9

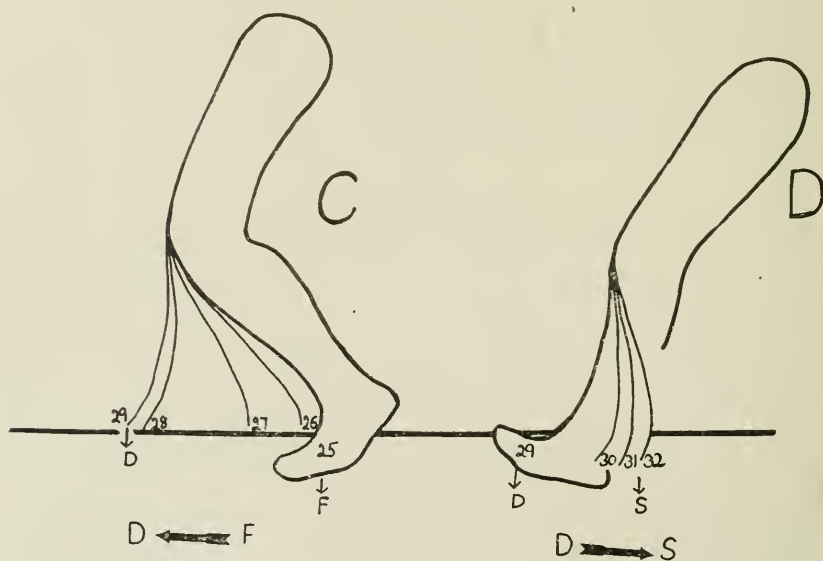


Figure 10

Figs. 9 and 10.—These illustrate the tetraphasic character of the knee movements. *A* and *D* constitute the diphasic posture element, *B* and *C* the diphasic flexion element. Only four tracings are shown in *D* as the next picture showed a position in the knee slightly in advance of its predecessor. This indicates a slight difference in the speed of walking or in the speed of taking the pictures. As a result only four pictures instead of five are needed to represent the last phase of posture in this particular pace. *A*, second phase of the support element; *B*, first phase of the flexion element; *C*, second phase of the flexion element; *D*, first phase of the support element.

Compare this statement with the data of Figures 1 and Table 1.

This illustrates how misleading and confusing grouping of muscle movements, as such and as they appear in certain reflexes, may become. The following quotation from Mackenzie⁶ gives a different and sounder point of view, but one which leads to confusion when applied to Walshe's terms.

The front of the leg and dorsum of the foot belong morphologically to the general extensor surface of the limb, corresponding with the back of the forearm and the dorsum of the hand. The back of the leg and sole of the foot correspond with the front of the forearm and palm of the hand, so that what we call flexion is really overextension. It is usual, however, and it will be followed here, to use the term flexion for the movement by which the dorsum of the foot is bent toward the front of the leg; the extension for the movement by which the sole of the foot is directed toward the back of the leg.

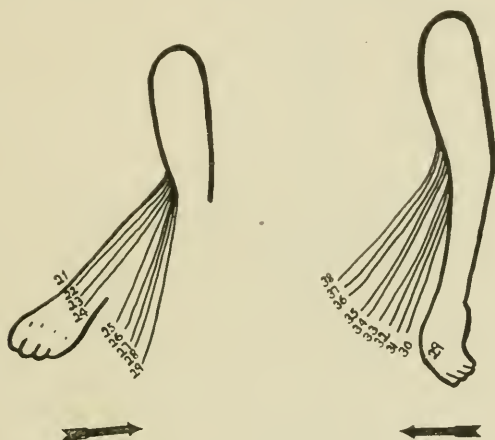


Fig. 11.—This picture illustrates the movements at the elbow as shown by the arrows.

Numerous other similar quotations could be given.

Movements of the Knee.—If a series of tracings of moving pictures is analyzed as far as movements of the knee are concerned, it is found that at the stage when the hip has ceased its backward or ventral movement, the knee is in direct alinement with it (Fig. 13). In the next tracing the hip starts to move forward or dorsally, while the knee starts to move backward or ventrally. This illustrates two points: 1. The ventral position of the leg, as has been previously contended, is one which leaves entirely out of consideration the knee and ankle and controls the leg as a rod. 2. Superimposed on this is the flexion

6. Mackenzie, W. C.: *The Action of Muscles*, New York, Paul B. Hoeber, 1918.

phase which begins with the process of carrying the leg forward—dorsally. When the ventral movements of the hip have ceased, that is, when the hip starts to move forward, or dorsad, if the movements at the knee are successively traced, it is found that the flexion reflex is at its maximum at a point of time midway between the assumption at the hip of the ventral and dorsal positions, as would be expected (Figs. 9 *B* and 10 *C*). As this point of maximum flexion reflex state is passed, the knee begins to extend until, in the dorsal position, it is again in alinement with the hip, producing a rodlike appearance (Fig. 10 *C*). When the successive stages between the forward (*dorsad*) and the backward (*ventrad*) positions are traced (Figs. 10 *D* and 9 *A*), that is, passing from dorsal through support into ventral, it is found that there is a little flexion at the knee at a point midway between the

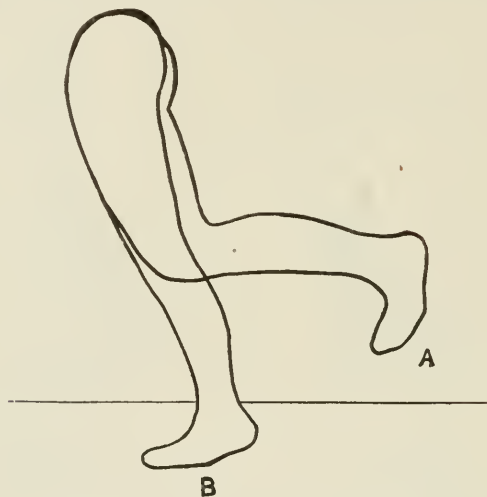


Fig. 12.—This illustrates the change in the location of the posterior thigh line, owing to contraction of the hamstring group of muscles, *A*, and indicates that the anterior thigh line must be used as a constant in comparing successive tracings of the thigh. *A* shows the contour in flexion at the knee; *B*, the contour in extension at the knee. ' "

dorsal and ventral positions and corresponding to the maximum flexion in the other half of the pace. In the sequence ventrad-dorsad it is also found that the hip of the leg of support is directly opposite the hip of the leg of progression (Fig. 5). Thus it appears that:

1. The movement at the hip is diphasic and passes from the ventral to the dorsal position with almost pendulum-like regularity (Figs. 7 and 8).

2. The movements of the knee are tetraphasic. Two knee phases occur during each hip phase (Figs. 9 and 10).

3. The knee phases occurring during the passage of the hip from ventral to dorsal (Figs. 9 *B* and 10 *C*) showed an excursion about twice as great as those occurring during the passage of the hip from dorsal to ventral (Figs. 10 *D* and 9 *A*).

4. On account of its anatomic make-up, though the actual movements carry the foreleg dorsad, the knee never goes into a truly dorsal position.

Flexion of the Knee in the Position of Support.—Furthermore, an unexpected finding has been noted, namely, that the knee is slightly flexed at the phase of maximum support (Figs. 5, 9 *A* and 10 *D*), corresponding to the flexion phase in the other leg and to a phase

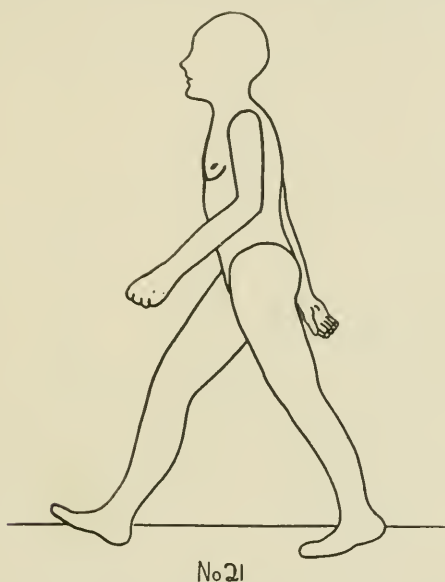


Fig. 13.—This illustrates the ventral position (left leg) and the dorsal position (right leg)—the first two elements of walking. Compare with Figures 5 and 14.

midway between the dorsal and ventral hip positions. If the particular tracing in which this is found is examined, it is noted that there is forward tilting of the torso, that is, that the center of gravity is not the same as in standing but is thrown slightly forward (Figs. 7 and 8). This slight flexion at the knee in the stage of support appears to be an adaptation to the forward placing of the center of gravity. As expected, in running, when the center of the gravity is still further forward, flexion of the knee in the phase of support was observed to be still greater. The oscillations of the knee into this slight stage of flexion (Fig. 10 *D*) and out of it (Fig. 9 *A*) are very much smaller

than those which occur in the flexion phase while passing from the ventral to the dorsal positions (Figs. 9 *B* and 10 *C*).

In the analysis of reflex stepping by Sherrington, previously alluded to, it was stated that in the "extensor" or antigravity phase (support), the knee was extended. In normal standing, this is the case. However, in the process of moving forward the pushing forward of the center of gravity somewhat negates this standing position. The importance of this in analyzing the condition known as decerebrate rigidity or antigravity posture, whether produced experimentally in animals or as a result of disease in man, is considerable; for, whereas the position of the legs in standing represents that reflex which Sherrington calls "the static of erect posture," the positions in walking and running represent the static posture plus a reaction to the forward placing

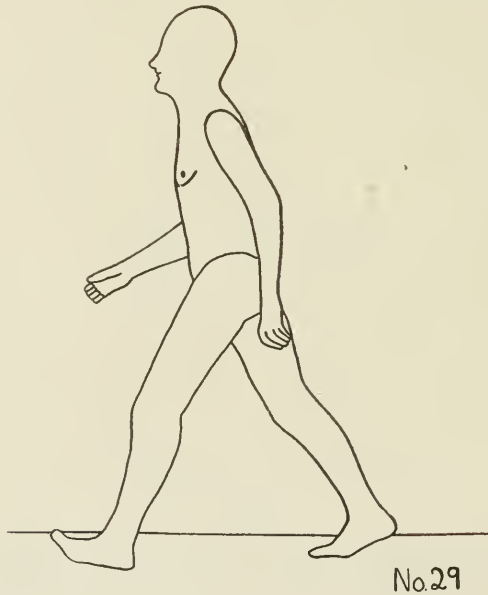


Fig. 14.—This illustrates the ventral position (right leg) and the dorsal position (left leg)—the first two elements of walking. Compare with Figures 5 and 13.

of the center of gravity. The importance in accounting for the flexion of the knees in the posture of parkinsonian syndromes has been alluded to in another article on that subject.⁷ The importance of this point in relating postural defects of hemiplegia, pseudobulbar paralysis and the parkinsonian syndromes will be discussed at length another time.

7. Kraus, Walter M.: An Interpretation of the Posture of Parkinsonian Syndromes in Terms of the Neuromuscular Mechanism, New York State J. M. **22**:9, 1922.

However, here it may be remarked that the unilateral defect of the person with hemiplegia represents involvement of the appendicular muscles only. No change of the torso in relation to the center of gravity occurs since the axial muscles having to do with this are not affected. When, however, bilateral involvement occurs, as in the parkinsonian syndrome and pseudobulbar palsy, the axial muscles are affected and the torso bends forward. As a consequence and as a reaction to the forward placing of the center of gravity, flexion at the knee analogous to that seen in the phase of support in walking appears. That group of neurons or integrations of the nervous system which maintains erect posture is apparently affected. The position of the leg in these three conditions is therefore primarily the same. Secondarily, flexion at the knee occurs in bilateral parkinsonian and pseudo-bulbar syndromes, due to the bilateral involvement.

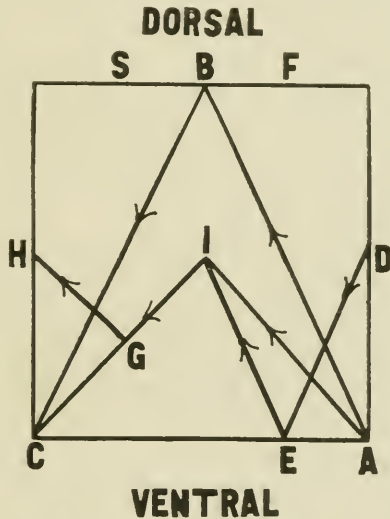


Fig. 15.—Graphic representation of the movements at the hip, shoulder, knee and elbow on one side of the body during a pace of a normal man. *A, B, C*, shoulder and hip. The movement begins at the ventral position, goes to the dorsal position and returns—diphasic; *D, E, I*, flexion phase at the knee—diphasic; *I, G, H*, support phase at the knee—about one-half the extent of *D, E, I*—diphasic. Note that these movements are all from a neutral position midway between ventral and dorsal, into a ventral position and out of a ventral position, back only to a neutral position midway between ventral and dorsal. The knee movement in a complete pace is tetraphasic; *A, I, G, C*, flexion movement at the elbow—diphasic. This shows the same limitations to the ventral field as *D, E, I* and *I, G, H*, the knee movements. The hip and shoulder movements are represented by one line, indicating that though apparently opposite in direction they are really, from a neuromuscular point of view, identical. *F*, maximum flexion; *S*, maximum support.

The position of the arm in the parkinsonian syndrome and in pseudobulbar palsy may easily be shown to be, from the neuromuscular point of view, the same as that of typical contracture in hemiplegia. This may be demonstrated by assuming the position of the arm in the parkinsonian syndrome and then successively contracting all those muscles which have produced this position, namely, the ventral group.

From this it may be seen that the entire hemiplegic position, from the point of view of posture or attitude, represents half of the position of the parkinsonian syndrome and pseudobulbar palsy, minus the reaction at the knee, which, in these latter diseases, is due to the forward placing of the center of gravity.

Identity of Homolateral Movements of the Arm and Leg.—In the earlier parts of this paper it was indicated that by means of the present method of interpretation of movements by the neuromuscular mechanism, it can be shown that the homolateral movement of the arm and leg represents the activity of the corresponding groups of muscles. Reference to the table will show that the leg is carried backward at the hip by a ventral group of muscles. Duchenne⁸ has shown that the gluteus maximus does not participate in either walking on the level or standing. This old observation, which I have been able to confirm, forms a final proof that the backward movement or extension at the hip in walking, is entirely ventral.

The forward movement of the arm is carried out by the pectoral muscles, also a ventral group. Thus it is seen that when the leg is carried backward and the arm carried forward both movements are brought about by ventral muscles. When, on the other hand, the leg is carried forward and the arm backward, both movements are carried out by dorsal muscles (Figs. 13 and 14).

At the elbow there is a ventral and a dorsal swing which coincides with the ventral and dorsal swing at the shoulder. The movements are diphasic (Fig. 11) unlike those of the knee, which are tetraphasic (Figs. 9 and 10). The shoulder movements are diphasic like those at the hip.

Figure 15 represents the most important observations of this article in graphic form.

V. SUMMARY

Analysis of the phases of normal human walking, in the light of knowledge of the embryology of muscles, their grouping and nerve supply, has shown that the customary interpretation based on ordinary observation of muscle actions is, in many respects, incomplete and incorrect.

8. Duchenne, G. B.: *Physiologie des Mouvements*. Paris, 1867.

The homolateral arm and leg, though apparently moving in opposite directions, are moved by the same group of muscles, ventral or dorsal. From the point of view of control by the nervous system, these apparently opposite movements are identical.

The phases of gait are four: (1) the ventral finlike movement of the lower limbs at the hip, (2) the dorsal finlike movement of the lower limbs at the hip, (3) the flexion reflex element, and (4) the support element.

The sequence of these is rhythmic. The order of sequence is 1, 3, 2, 4—1, 3, 2, 4, etc. The first two (1 and 2) represent activity of singly hinged appendages. The second two (3 and 4) represent activity of three-hinged appendages. The first two and the second two each constitute a pair of corresponding opposites. The two elements of the second pair correspond to the flexor (3) and the extensor phases (4) of reflex stepping as described by Sherrington.

In the first two the leg acts like a rod, leaving out of consideration the two large joints at the knee and ankle. The muscular activity of the ventral phase is obviously antagonistic and opposite to that of the dorsal. The alternating muscular activity at the three great joints of the flexion reflex phase is also opposite to that of the support phase.

Analysis of the hip movements shows them to be diphasic. The shoulder and elbow show the same diphasic activity as the hip. Analysis of the knee movements shows them to be tetraphasic. Two phases occur during each hip phase. The two hip phases are from the ventral to the dorsal position and from the dorsal to the ventral position. The two knee phases constituting the flexion reflex element of a pace occur in the ventral to dorsal hip phase. The two knee phases constituting the support element of a pace occur in the dorsal to ventral hip phase.

In the flexion reflex phase the knee passes through an arc about twice as great as in the support phase. At the maximum support stage the knee is slightly flexed. This is due to the forward placing of the center of gravity of the body.

In the typical postural defect of hemiplegia only the appendicular muscles on one side are involved. In the typical bilateral postural defect of parkinsonian syndromes and pseudobulbar palsy the appendicular muscles are involved on both sides, as well as the axial muscles on both sides. The facts given in regard to the forward placing of the center of gravity show that the typical postural defect of hemiplegia represents half of the typical bilateral postural defect of the parkinsonian syndrome and of pseudobulbar palsy, minus the reflex to the forward placing of the center of gravity.

THE NATURE OF CERTAIN FUNCTIONAL NERVOUS DISTURBANCES AND THEIR TREATMENT ALONG METABOLIC LINES *

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It is my purpose to attempt to outline some of the pathologic conditions concerned in certain functional and possibly a few organic disturbances within the field of neurology; and to direct attention to some forms of treatment based on these.

In the course of observation on about 1,000 cases of arthritis, a great many associated clinical disturbances were found. Among the chief of these, so far as the neurologic field is concerned, are fatigue, mental lethargy, headache and migraine, vertigo, tinnitus, neuritis, practically the whole complex of neurasthenia in its many phases and mental depression. In the process of treatment of the arthritic patients, many of the associated neuropsychiatric symptoms disappeared wholly or in part. It is obvious, therefore, that the same underlying causes exist in both groups to a large degree.

To focal infection as the cause of disease has been ascribed too generic a rôle, but there is no doubt that a large number of functional neuropsychiatric disturbances have been shown definitely to arise from such premises.

In considering so-called focal infection and its consequences, however, it is important to realize that a considerable variety of agents or conditions may act in an analogous manner and produce the same results. This broad truth is inadequately appreciated. It was evident, for example, during the studies on arthritis, that in addition to the accepted focal infections within the mouth, head, genito-urinary tract and elsewhere, many diseases, especially dysentery, acted comparably. Exposure to cold and wet was productive of the same consequences and, indeed, constituted the largest immediately precipitating factor. It is important, therefore, to appreciate that the disease processes arise from any interruption in a fairly long chain of established physiologic events, and that even mechanical disturbances of certain broad anatomic relations may contribute to this end.

This chain involves various mechanisms, including the large field of the respiratory functions of the blood, now being subjected to so much study; the ductless glands, particularly the thyroid; the metabolism of

* Read by invitation before the Philadelphia Neurological Society. Nov. 24, 1922.

the muscular system; the gastro-intestinal tract; and a large number of accessory physiologic acts which normally preserve the balance of health, such as exercise, pulmonary ventilation, perspiration, and the like. There is no opportunity here to discuss at length the dynamic pathologic condition caused by interruption of this chain; but it may be said that there is reason to regard it as arising from interference with oxidative metabolism, using that term in a wide sense.

Some of the reasons for this view are to be seen in the results of the studies on arthritis and focal infection referred to in the foregoing.¹ Omitting negative data which helped to delimit the problem these results were, in brief: a slight lowering of the basal metabolism in 20 per cent. of the cases studied; a high value for blood creatin in about one half of the cases studied, which can be interpreted as evidence of disturbance in the metabolism of carbohydrate; a slight lag in the elimination of water and salt, and a lowering of the sugar tolerance more or less proportionate to the severity of the arthritis.

This lowered tolerance tends to return to normal with convalescence or recovery, but does so most abruptly after the removal of apparently causative focal infection. The contrast under these circumstances may be striking.

It is important to realize that a lowered sugar tolerance is not specific for any disease, even diabetes, and is to be regarded somewhat in the light of a leukocytosis or fever chart. Inasmuch as a lowered sugar tolerance seems to be a function of inflammatory processes at large, it accompanies a considerable range of conditions, and has been made the basis of some too sweeping conclusions. If these limitations and absence of specificity be borne in mind, however, the test becomes valuable for reflecting changes in the so-called dynamic pathology which morphologic methods of study do not reveal.

Starting from this premise, B. M. Hendrix, C. Y. Crouter and I, in collaboration with R. B. Osgood,² observed in a long series of normal, arthritic and variously diseased subjects under certain conditions of study, that parallel with the lowered sugar tolerance which accompanies severe expressions of focal infection, there was generally a rise in the percentage saturation of the blood with oxygen. This rise was not reflected in normal subjects, whose sugar tolerance was normal. Furthermore, arthritic patients as a group showed a slightly higher percentage saturation of their venous blood with oxygen than did normal persons or certain other types of diseased subjects; and in some cases

1. Pemberton, Ralph; Robertson, J. W.; Tompkins, Edna H.; Foster, Goodwin L., and Buckman, Thomas E.: Studies on Arthritis in Army Based on Four Hundred Cases, *Arch. Int. Med.* **25**:231, 241, 243, 335 and 351, 1920.

2. In process of publication in the delayed September number of the *Journal of Metabolic Research*.

the figures were very high. This high saturation may remain roughly constant over long periods of time. In the light of these data, it is reasonable to deduce that something interferes with the removal of the oxygen from the venous blood as it passes through the tissues. On a number of counts, such as study of the dissociation curves of the blood for oxygen and carbon dioxide, the mechanism does not seem to be chemical in nature, and the thought presents itself that the explanation lies in the rate at which the blood passes through the tissues owing to some change in the peripheral vascular mechanism. This has not been proved as yet, though arthritic patients seem to have a lower rate of blood flow in the hands than do normal patients, as measured by Stewart's method of calorimetry. The real *modus operandi*, therefore, must await further investigation. It should be stated, as Meakins and Barcroft have pointed out, that certain external conditions, such as great heat locally, may somewhat similarly modify the blood gases in the same direction; but these are not adequate to explain the foregoing findings. Coupled with a slightly lowered basal metabolism and the lowered glucose tolerance, these facts, together with a considerable number of clinical data, strongly suggest that the underlying difficulty relates to some part of the oxidative equation; by this is meant the removal of the products of oxidation as well as the delivery of oxygen to the tissues.

The exhaustive studies conducted on aviators in this country and Europe during the war have demonstrated that some of the symptoms above described, such as headache, mental lethargy, fatigue and variously confused mental states, can be brought about by exposure to conditions of low atmospheric pressure. These experiments are conducted under conditions which simulate, so far as the gases of the blood are concerned, those encountered at high altitudes, the chief factor in which is the anoxemia produced by the lessened amount of oxygen available.

It is interesting to note as a corollary to these findings that the agents which are of most use in combating the effect of focal infection, apart from operative removal of it, are those which in one way or another stimulate metabolism. Indeed, it is frequently possible to observe benefit from some of them to the neurologic complications which accompany arthritis or which may arise, independently of it, from the same causes. This has been particularly noteworthy in relation to headache, mental hebetude and neuritis. The agents in general which are helpful are hydrotherapy, exercise, massage, arsenic, the iodids, radium, thyroid extract, nonspecific protein and some others. In one way or another, these depend for their effect on closely comparable actions, many of which can be demonstrated in the precise terms of the laboratory. The increased metabolism of arsenic and radium can be observed in the urine; and the influence of exercise,

hydrotherapy and thyroid extract is easily demonstrable through calorimetry. Much of the relation of the endocrins to the syndrome under discussion is speculative, but so far as the thyroid is concerned, it is in some part at least exact; and Kendall has adduced evidence to show that thyroxin acts by virtue of removal of the carbon dioxid picked up by one of its benzene rings.

In view of the wide use of hydrotherapy made by alienists in the form of continuous immersion in the hot bath, it is of interest to note the results of some studies by C. Y. Crouter and myself³ on the response of the body to external heat in the form of electric "bakes." It was found that during the bake the sweat almost invariably changes its hydrogen-ion concentration or "reaction," whatever this may have been at the start. It is generally about neutral at the outset, but becomes more alkaline or less acid as the bake progresses. The most striking exceptions to this have been among arthritic patients whose sweat was more acid than that of any normal person and sometimes showed no changes of reaction, remaining at the same acid point throughout.

It may be added further that if an arm is surrounded by a rubber bag during a "bake," determinations of the carbon dioxid of the air in the bag show that this increases coincidentally with the change in the reaction of the sweat. Whether this is due solely to the increased metabolism of the sweat glands or to an increased elimination of carbon dioxid independently of this, cannot now be stated, but the end-result is an added loss from the body of the chief end-product of combustion. The question is under further investigation. There are other changes of metabolism of much interest bearing on the subject of the therapeutic application of external heat, whose technical nature does not allow of inclusion here.

We thus have two sides to the picture: On the one hand, there is evidence of impaired oxidative metabolism, and on the other, evidence of benefit from the agents which improve this mechanism.

There is still another way by which the body metabolism may be importantly modified. Metabolism is divided roughly into two kinds: endogenous, or that which goes on irrespective of material introduced into the economy; and exogenous, or the metabolism of that which is introduced from without, such as food, with its subsequent reciprocal influence on the body tissues. The therapeutic measures and agents mentioned influence chiefly the endogenous metabolism, although perhaps all phases are affected. The exogenous metabolism is, by definition, open to great modification by changes in the dietary, and there is in this another means of influencing the oxidative equation above outlined.

3. Pemberton, Ralph, and Crouter, Caroline Y.: The Response to the Therapeutic Application of External Heat, J. A. M. A., to be published.

As previously mentioned, development of the value of the dietary to the syndrome under discussion originated in relation to arthritis, but enlarging clinical experience has shown its applicability to wider fields. There are several ways in which it operates. If the syndrome under consideration is characterized by difficulty in completing the oxidative processes, broadly speaking, it would seem logical that attempts to modify the burden imposed on this mechanism might have a useful influence; and, indeed, experience has shown this to be the case. In general, the benefits from diet under these circumstances are obtained through lightening this burden, that is to say, chiefly through reducing the caloric total of the food ingested. There are differences in the way in which the three foodstuffs act, but whatever the proportions of the foodstuffs, within reasonable limits, the result will be much the same if the total number of calories is reduced. Of the three foodstuffs, protein, carbohydrate and fat, reduction in the carbohydrate is followed by the largest benefits, probably because this substance yields under average conditions, the largest quota of calories, and hence is most concerned in the combustive processes. It is, perhaps, not generally appreciated by those working in fields unrelated to metabolism that nearly 60 per cent. of the protein food eaten becomes carbohydrate within the economy and is combusted in this form. It can be seen from this that often there may be advantage also in reducing the protein intake, though not for the reason popularly accepted that "red meats" in themselves are injurious. There is little or no evidence to indicate that the nitrogenous fraction of the protein molecule contributes importantly or specifically to the pathologic condition mentioned in a strict metabolic sense.

The rôle of the fats is less easy to define, but it is probable that they act in about the same way, although their slower rate of combustion sometimes permits of their larger utilization.

TREATMENT

Treatment, by means of dietetics, of the metabolic disturbances arising out of the agents and conditions mentioned, consists, therefore, essentially in catering to a weakened function, and presents the advantages and limitations of such a procedure. When the reserve of the organism is adequate, lightening the load is sometimes followed by brilliantly successful consequences. If function is much impaired, however, these results may be produced slowly; they may be produced only when abetted by measures which specifically improve metabolic function; or they may not be produced at all.

The situation is in some ways comparable to that occurring in cardiac decompensation. It is obvious that an overtaxed heart may respond to rest alone; or it may be beyond benefit from the lowered demands which

rest alone determines, and may then require further and accessory therapy from digitalis and other measures; or, finally, it may not respond at all to even a combination of measures. The analogy to cardiac decompensation is not complete, since rest is probably always harmless; whereas adjustment of the metabolic burden beyond a certain point may entail varying degrees of undernutrition.

It should be emphasized at this point that treatment of a disease-process on the basis of adjusting a burden to a weakened function should rarely be carried out if the cause of that weakened function can be removed; or in the presence of anemia, marked asthenia, undernutrition, and the like. The very definition of such therapy presupposes that the removal of a burden is accompanied by a capacity to react after such removal. This cannot be too strongly emphasized. There is nothing in these measures which in any way contraverts the important principles of rest and forced nutrition, when indicated, and, indeed, the factor of rest assumes even added importance.

It is my custom, in attempting treatment along this line, to determine the average caloric intake for a given person. This usually requires the cooperation of a dietitian in weighing food and estimating caloric values, but from such data there can be obtained a visualization of the burden on the exogenous metabolism. In instances where this is great, because of a large intake of food with a high caloric total, the metabolism can be spared a large burden by reducing this without detriment to nutritional needs. Thus, if the accustomed intake is 3,000 calories, it is obvious that 1,000 calories can be omitted with entire safety under average conditions. If, on the other hand, the accustomed intake is 1,500 or 1,800 calories or less, as is frequently the case in chronic invalidism, further curtailment must be cautiously made with the fullest regard for nutritive necessities. Complete starvation, accompanied by conditions approximating basal metabolism, is sometimes followed by striking results, but I desire to urge caution against the lax use of such measures. The proper selection of the type of cases and the proper conduction of the measures concerned are the *sine qua non* of success. Space will not permit me to cite illustrative cases which have been detailed at length in previous publications.⁴

There is another mechanism by which curtailment of diet may have gratifying results. It has been clearly shown by many workers, chiefly in surgical fields, that the colon often plays an initial rôle in causing disease by virtue of absorption of various evil products. Irrigation has been depended on in large measure to further elimination of this material; and while this is useful, it is common knowledge that it often

4. Pemberton, Ralph: The Use of Diet in the Treatment of Chronic Arthritis. *Am. J. Med. Sc.* **161**:517 (April) 1921.

fails. In this connection, it is pertinent to note that the materials which it is desired chiefly to remove by irrigation are precisely those which are in large part ingested by mouth, and that in cutting down on the dietary we also cut down on the substances producing these undesirable end products. The foodstuffs which give rise to them within the colon are partly, but not exclusively, those which are susceptible to putrefaction, namely, highly nitrogenous foods; and in cases presenting this difficulty, there may be real advantages in withholding such substances as meat and eggs. Carbohydrate foodstuffs may behave similarly, however. On broad grounds it can be easily seen that efforts to meet such a pathologic condition by flushing out the colon is something of an Augean task unless the supply is controlled at the source, a fact which is widely overlooked.

It is not to be supposed that the measures last mentioned, or, indeed, any measures directed toward the neurasthenic syndrome, have universal applicability or that they act as panaceas. If the general nature of the pathologic disturbances under consideration be borne in mind, however, it may be appreciated that the various measures discussed have their respective, though limited, applications, and that results may often be achieved by combining them, where dependence on one measure alone would fail. It is for this reason that the results of correcting or removing "focal infection" are so often disappointing. It is to be appreciated that following long-standing mal-function of various chronic diseases and long-standing focal infection, there may be a more or less permanent dislocation of many normal physiologic processes, which may require time and many accessory measures for restoration. The orthopedists have brought to bear a helpful point of view, in certain cases handicapped by faulty posture or "bodily habit"; but in severe cases restoration by any means may be impossible.

ILLUSTRATIVE CASES

Illustrations of immediate and delayed return to normal of the disturbed physiology described can be seen, for example, in such instances as the following: A soldier of 19 suffering from an arthritis of nine months' duration presented badly infected tonsils and a greatly lowered sugar tolerance. Two weeks after removal of his tonsils his clinical symptoms had entirely cleared up, and the sugar tolerance had returned to normal. Another example was that of a young soldier suffering from an arthritis of the hands and feet. About two weeks after removal of his tonsils, which were badly infected, his clinical symptoms had cleared up, but the lowered sugar tolerance had not fully returned to normal, although it was greatly improved.

A man of 40, presenting spondylitis and abscessed teeth, showed improvement in his general condition and improvement in his lowered

sugar tolerance following attention to these foci, but the sugar tolerance remained at an abnormal level for many weeks.

Finally, several cases which had been watched critically during complete convalescence along the line of restricted diet showed a slow though extensive return of the sugar tolerance to normal. In some patients who recovered after this treatment, the lowered tolerance persisted at its previous level. It is fair to deduce in the latter cases that improvement by this form of treatment was achieved by catering to a lowered function and placing lessened demands on it. The function remained disturbed, but was able successfully to perform smaller amounts of work. Disturbances in the blood gases described can be shown sometimes to follow the same general course.

So fundamental are the changes in metabolism brought about by the various measures discussed that I am prepared for wider application of them than has yet been made. The more fundamental the physiology concerned, the wider the consequences of its disturbance and correction. The processes of oxidative metabolism are essentially the same in whatever tissue they occur, whether nervous, muscular or glandular, and are dependent on the same underlying factors, though their expressions in terms of disturbed function vary greatly.

METABOLISM IN THE NERVOUS SYSTEM

It is of the highest importance to appreciate that the various tissues of the nervous system have an active metabolism in which the processes of utilization of oxygen and production of carbon dioxide and heat are real and measurable. According to Mathews,⁵ the need for oxygen is greater in the nervous tissues than in any other tissue of the body. Study of the nervous system in its more precise aspects has been confined almost exclusively to histopathologic and anatomic observations. Students of metabolism, in general, have focused their attention on other and more accessible structures, but it is none the less true that the nervous system, like every other tissue, is dependent on processes of true combustion accompanied by the synthesis and disintegration of tissue. The thoroughness and facility with which these processes are carried out condition the efficiency of the cell unit. A host of factors influences these processes, and the cells in nervous tissue may be laboring under a heavy burden although giving little or no evidences of that to histologic study. These disturbances may arise quite apart from trauma or infection, although it is doubtless true that infection of various kinds operates the more easily on such prepared soil. The work of Child⁶ in relation to reestablishment of the youthful type of met-

5. Mathews: *Physiological Chemistry*, Ed. 4.

6. Child: *Am. J. Physiol.* **48**:256.

abolism in planaria following starvation, is full of suggestion and, in conjunction with other work along similar lines, corroborates the point of view outlined above.

In few instances are disease processes confined to isolated metabolic errors, as was formerly more widely believed. Normal physiology provides for a considerable margin of substitution and addition in its processes, so that one system can often function somewhat in lieu of another or accessory to it. Though the chain of normal events is long, interruption at one point may be entirely compensated for; but, if not, correction must in general depend on overuse or development of other parts with proportionate adjustment of burdens elsewhere. This is the lesson being taught by the study of diabetes, rickets and the interesting fields of the acidbase equilibrium and the respiratory functions of the blood.

The more precise laboratory methods which sometimes reveal the pathologic disturbance under discussion may occasionally be used for detecting this disturbance when the superficial clinical manifestations of it are slight. Thus, a lowered sugar tolerance or a disturbance in the blood gases, as above outlined, a high blood creatin, and possibly changes in the reaction of the sweat, may sometimes be of diagnostic value in doubtful cases, and may also be used at times as criteria of the return of this dislocated function to normal. It would be improper to place undue emphasis on the information to be derived from this source, but under certain circumstances it may be very real.

SUMMARY

Many forms of neurasthenia, headache, migraine, most cases of neuritis, certain types of mental depression and melancholia—possibly a few supposedly organic conditions—are the result of interruptions in a chain of metabolic processes.

Part of the metabolic process apparently concerned is, broadly speaking, oxidative in nature. By this is meant the delivery of oxygen, the removal of the products of combustion or both. Interruption of the normal course of events may be due to a large number of factors, among which are external physical conditions, the endocrine system, focal infection, mal function of various viscera and various metabolic insults. Appreciation of the general nature of this disturbance makes possible the stimulation of metabolic functions by a variety of measures on the one hand, and adjustment of the metabolic load on the other. A combination of both measures is sometimes helpful or necessary.

Among the few methods of adjusting the metabolic load is that afforded by diet. When applied to properly selected and appropriate cases important results may follow its use. The limitations and indi-

cations in employing it must be critically borne in mind, and at least a working knowledge of nutritional problems is necessary to safety and the best results.

Some of the laboratory findings, such as a lowered sugar tolerance, changes in the blood gas equilibrium, increased values for blood creatin and possibly changes in the reaction of the sweat, accompanying the syndrome under discussion, may, in the absence of a clear clinical picture, serve at times as indexes of the pathology concerned.

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Abstracts from Current Literature

THE SYNDROME OF THE CENTRAL GRAY NUCLEI. GEORGE BICKEL,
Rev. méd. de la Suisse Rom. No. 6: 365 (June) 1922; No. 7: 434 (July) 1922.

Of the gray masses of the cerebrospinal axis, there are none about which we know so little as the central gray nuclei. Only during the last fifteen years has any advance been made in the study of the function and disturbances of these basal ganglions. This advance has been brought about especially by the work of von Monakow and Déjerine. Before beginning this study the author reviews the anatomy of the basal nuclei, this being of importance for the interpretation of the phenomena consecutive to their alteration.

Anatomy of the Central Nuclei.—The author divides the central nuclei into three groups: (1) thalamic nuclei, (2) caudate, (3) lenticular. The two latter are commonly known as the corpus striatum. A study of the optic thalami shows that they are in reality formed by several nuclei, more or less completely fused at certain points. The subdivision of Burdach is the one usually employed—the anterior, the external and the internal nucleus. The external nucleus is called the pulvinar, and by some is considered independent of the optic thalamus. The external geniculate is also considered separate from the pulvinar. Between the external and internal nuclei is a fourth (Luys' nucleus), and finally a fifth, below and in back of the fourth, called the semilunar nucleus of Flechsig. Von Monakow has developed a more minute classification, each of the above nuclei having separate subdivisions, and each division having a cerebral representative. Histologically, the optic thalami are made up of two types of nerve cells; the one with long axis cylinders and the other with short axis cylinders.

The corpus striatum is composed of two separate nuclei, the caudate and the lenticular. The lenticular nucleus is divided into two parts by bands of white fiber. The external portion is called the putamen; the two internal portions, the globus pallidus. The researches of Bielschowsky have shown that the globus pallidus is composed of cells of type 1 of Golgi. The putamen is made up of two types of cells, some type 1, but most small cells of type 2 of Golgi. The caudate nucleus has also two types of cells similar to those of the putamen, with which it fuses in its inferior portion.

In place of the division of the corpus striatum into lenticular and caudate nuclei, recent work tends to the classification of the centers on a histologic basis. The caudate nucleus and external portion of the lenticular nucleus or putamen are identical and are now called the striatum or neostriatum, while the globus pallidus is called pallidum or paleostriatum.

Taking up the connection of the various ganglions between themselves and between the remainder of the brain, the author states that:

I. Union of Central Gray Nuclei Between Themselves: 1. The striatum is connected to the pallidum by the striopallidal fibers. Experimental destruction of the putamen in monkeys, as done by Wilson, shows a degeneration of these fibers, the degeneration going no farther than the pallidum. 2. The thalamus furnishes the only afferent fibers to the striatum. Destruction of the thalamus in monkeys causes a secondary degeneration of these fibers. 3. The pallidum unites with the optic thalamus by a system of afferent fibers, which after traversing the external capsule run to the external side of the thalamus.

II. Union of Central Nuclei with Cortex: 1. The thalamus connects with the cortex through the corona radiata, which also has the corticothalamic fibers coming from all portions of the cortex. 2. It appears established that there is no true connection between the corpora striata and the cerebral cortex.

III. Union of Central Nuclei with Subthalamic Centers: 1. The thalamus connects with the subthalamic regions by three groups of fibers: (a) with the nuclei of Goll and Burdach through the tract of Reil, (b) with the opposite olives through the superior cerebral peduncle, (c) with the red nucleus and the reticular formation of the cerebral peduncles by the fasciculi of Forel and the radiation of Calot.

There are also connections between the optic thalami and the anterior corpora quadrigemina and the visual apparatus by fibers between the pulvinar and the external geniculate body. There are analogous communications between the nuclei amygdalae and the area olfactorius by the taenia semicircularis; with the mamillary bodies by the fasciculus of Vicq d' Azyr; with the ganglion of the habenula by the olfactory radiations and the fasciculi septothalamici. In most of these connections there are fibers which place the optic thalamus in relation to the motor system by the way of the pyramidal tracts. The existence of these fibers is still doubted.

2. Fibers descend from the corpus striatum in four groups: (a) fasciculus pallidorubral, ending in the red nucleus; (b) fasciculus pallido-Luysii, ending in the hypothalamus; (c) fasciculus pallidonigri, going to the locus niger; (d) striotegmental fasciculus, to the nucleus of the posterior commissure and to the nucleus of Darkschewitsch.

The optic thalamus, thus, has numerous connections with the cortex. The corpus striatum, on the contrary, is an organ strictly autonomous, independent of the cerebral cortex, its afferent fibers coming directly from the optic thalamus and the efferent fibers going either to the thalamus or to the subthalamic region.

According to Malone, the pallidum is made up of the motor type of cells; the thalamus, of the sensory type.

Clinical Anatomic Syndromes.—The thalamic syndrome is the best established syndrome of the basal nuclei. Associated with this are the striate and pallidum syndromes.

I. The Thalamic Syndrome: This syndrome was first noted by Déjerine and Egger in 1903. It can be determined best by the development of a tumor, hemorrhage or thrombosis. The symptomatology is usually unilateral, this being a point of distinction from the other basal nuclei syndromes, which tend to be bilateral. The chief symptom is a dissociated hemianesthesia, mild in the superficial sensations but profound in the deep sensibility. There is always an *anesthesia dolorosa*. The pain is continuous with paroxysmal exacerbations. It may be paresthetic in type (formications, engorgements, etc.), and rebels against all treatment. Hemiataxia occurs. This never attains the grade of a tabetic ataxia, and often must be searched for. The mechanism of the ataxia is due to a failure of the coordinating centers to measure with exactness the motor impulses necessary for the correct production of a motion. If the lesion is near the pulvinar, one frequently has an homonymous hemianopsia of the opposite side. This may also occur if the lesion is in the radiations of Gratiolet or in the external geniculate body. A mild hemiplegia may be associated, usually transitory, accompanied by choreo-athetoid movement. These symptoms may be due to involvement of the internal capsule or striate body. The thalamic syndrome is then a sensory syndrome, characterized by hemianesthesia, hemiparesthesia and hemiataxia.

II. The Strioglobular Syndrome: Under this heading are included: (1) Progressive lenticular degeneration (Wilson). This syndrome was described by Wilson in 1912, though reference had been made to this condition previously. The affection is usually familial, rarely occurring in two successive generations. It appears, as a rule, between the ages of 10 and 25; it progresses slowly, usually terminating in death in from two to seven years.

The characteristics are motor involvement consisting in involuntary movements and hypertonia. The involuntary movements manifest themselves in a tremor, which is the most constant sign. The tremor at first slight, becomes more marked, is rhythmical, most marked in the arms, but may affect all portions of the body. At rest the tremor may disappear; with voluntary movements it is increased. It is an intention type of tremor, similar to that in multiple sclerosis. Some observers describe choreiform or athetoid movements, which are not part of the picture as described by Wilson. It is difficult to say whether the latter signs are truly a part of the syndrome or due to adjacent phenomena.

Rigidity is the most important symptom. It begins insidiously, becomes more marked, appears first in the lower limb, and may spread to all portions of the body. With the increasing rigidity there are contortions, and the limbs and head are held in bizarre positions. Wilson pointed out certain peculiarities which distinguish this hypertonia from ordinary pyramidal spasticity. In ordinary spastic paralysis there are, as a rule, pyramidal signs, such as increased deep reflexes, the Babinski sign and others, all of which are missing in an uncomplicated Wilson's disease. There is also in Wilson's disease a peculiar rigidity of the head, especially of the face, giving the face an odd expression of stiffness and slowness of motion. In the limbs and body the hypertonia creates stiff attitudes, sometimes not unlike tetany. During rest the hypertonia disappears. Power is well preserved, but movements cannot be properly produced.

Usually the rigidity involves also the muscles of phonation and deglutition. The speech becomes altered, it is dragging, scanning and difficult. The muscles of phonation are movable; no trace of atrophy is found; but the patient is unable to talk because of the spasms which hinder him. At times the spasms cease entirely, and the patient may utter a few words intelligibly. Deglutition may be markedly impaired so that even liquids are swallowed with difficulty. Saliva accumulates in the mouth, and there is considerable drooling.

This trio of symptoms—difficulty in speech, deglutition and salivation—suggests a pseudobulbar paralysis. There is, however, no real paralysis, but only the spastic condition. These patients are also emotional; they cry and laugh easily. The gait is disturbed as are all movements. Late in the disease walking may be impossible.

Psychic disturbances are usually present. The patient is dull and slow, at times euphoric, much in contrast to the mental enfeeblement. As described by Wilson, the anatomic lesions are an extreme atrophy of the striatum and pallidum. There is bilateral softening of the lenticular nucleus, especially of the putamen, which may be cystic, leaving the globus pallidus unaffected. There is a destruction of nerve tissue with replacement of neuroglia; later the neuroglia degenerates and cysts are found. Outside of the corpus striatum the lesions are of little importance. The only degenerated nerve fibers are the striofugal fibers which go to the thalamus and the subthalamic nuclei. Wilson insists that the lesions are entirely in the extrapyramidal motor system. tracts going from the striate nuclei to the red nucleus and continuing into the

spinal cord by the fasciculus rubrospinal of Monakow. The sensory tracts and pyramidal system are always intact. A hobnailed liver is always associated with the cerebral pathology. The liver picture is especially interesting as it never causes the clinical picture of a cirrhosis. The spleen is hypertrophied. All other organs are normal.

This syndrome, according to the author, merits a special place in pathology and, as Wilson stated, is a new disease.

(2) The Pseudosclerosis of Westphal-Strümpell: This affection closely resembles Wilson's disease. It is characterized by gross trembling, speech disturbance and marked spasticity. It was first described by Westphal in 1883. It was not until 1912, however, that Hoesslin and Alzheimer described the lesions of the central nervous system. In summary, one has an affection characterized by a coarse tremor of the entire body, exaggerated by voluntary movements and by emotions. There is a muscular rigidity, less marked than in Wilson's disease, a gait and speech disturbance and mental changes which may progress to dementia. As in lenticular degeneration, there is a change in the liver and spleen. Finally, in all cases one finds a ring of brownish-green pigment at the periphery of the cornea, which does not exist, as a rule, in Wilson's disease. In the nervous system, one finds a glia proliferation with partial atrophy of the nerve structures, especially the ganglion cells. These alterations are especially noted in the basal ganglions, as the corpus striatum and hypothalamic region. Changes occur in the cortex, cerebellum and stem. There has been considerable discussion as to the classification of Wilson's disease and pseudosclerosis. The author discusses the various views but comes to no conclusion. As to the etiology of these conditions, little can be said. At present the conditions may be looked on as a constitutionally congenital anomaly, heredofamilial in type.

(3) The Syndrome Pseudobulbar Senilis: In connection with Wilson's disease, it is well to mention the pseudobulbar paralysis of the old, in which there is sometimes a bilateral sclerosis of the lenticular and caudate nuclei. The type of lesions and their location naturally provoke identical symptoms.

III. *The Partial Striate Syndromes.*—In certain conditions lesions are confined to either the caudate or lenticular nucleus, or to the caudate nucleus and putamen or pallidum. Their syndromes are classed as follows:

A. Striate Syndromes (Choreo-Athetoid Syndromes).

(1) Double Athetosis: This was first described by Anton in 1896. The credit for a clear description of the disease belongs to C. Vogt. The affection begins in infancy, is progressive, but usually becomes stationary about the eighth year; it may even become regressive. Clinically there is a bilateral hypertonia associated with athetoid movements, sometimes choreo-athetoid. The speech is of a pseudobulbar type, with spasmodic crying and laughing. In appearance it resembles Wilson's disease, but is distinguished by the athetosis, absence of tremor, its early onset, absence of cirrhosis and difference in cerebral lesions. There is an atrophy of the caudate nucleus and putamen (*état marbré du striatum*).

(2) Chronic Chorea (Huntington's): The clinical symptoms are well known, and divide themselves into two large groups—progressive mental deterioration and involuntary motor disorders. Jellgersma, in 1908, attributed the disease to an atrophy of the caudate nucleus, which was confirmed by Alzheimer in 1911. Marie and Lhermitte have done the most important work and established that the pathology is an atrophic degeneration of the corticostriate fibers. They found an atrophy of the caudate nucleus and putamen,

also an atrophy of the cerebral cortex, especially in the frontal and rolandic regions, and, finally, slight changes disseminated throughout the brain. The motor condition is thought to be due to the lesions of the striatum; the psychic manifestations, to cortical changes.

(3) Acute Chorea (Sydenham's): In acute chorea there is an extensive encephalitis. The chief damage occurs in the caudate nucleus. It is practically impossible to differentiate between the lesions of an acute and a chronic chorea.

(4) Chorea and Athetosis in Hemorrhagic Lesions and Softenings: Hemorrhages and softenings, when situated in the striatum, may cause choreo-athetoid movements. Such cases seem to be rare.

B. Syndrome of the Pallidum (Parkinsonian Syndrome).

Lesions of the pallidum have long been considered as the cause of paralysis agitans. It is now more or less accepted that the so-called Parkinson's disease is not a morbid entity, but a syndrome, the result of a variable etiology, and due to pathologic changes in similar portions of the brain. Clinically, the syndrome has three chief components: muscular rigidity, abolitions of automatic and associated movements, and a slow tremor which may involve any or all portions of the body, even the vocal cords. Sweating, salivation, paresthesias and cramps may occur. The psychic remains intact, although a certain exaggeration of emotivity may be present.

According to Souques, five types of parkinsonism are distinguished: (a) senile or presenile type (classic form), (b) juvenile type, (c) paralysis agitans without agitation, (d) parkinsonian syndrome due to hemorrhage or softening, (e) a postencephalitic Parkinson's syndrome.

While it is accepted that the chief pathologic changes occur in the globus pallidus, there is still some room for discussion; thus Tretiakoff is of the opinion that the chief changes occur in the locus niger. The former is, however, the accepted view.

With the foregoing review, the major syndromes of the lesions of the central nuclei have been described. In passing, the author mentions a few other syndromes but does not elaborate, stating that too little is known about them: disintegration and dysmyelination of the stria (Vogt), dystonia lenticularis of Spiller, arteriosclerotic muscular rigidity (Försler), pyramidopallidal degeneration syndrome (Lhermitte and Cornil), spasmodic pseudosclerosis (Jacob).

Pathologic Physiology.—While the clinical anatomic experiences have tended in a measure to clear away some of the mystery surrounding the central gray nuclei, experimental physiology has tended to complicate the picture. Unfortunately, all experimental results have been so variable and the problem so difficult that the results thus far have been unsatisfactory.

General Conclusions.—In conclusion, the author states that the symptoms of the thalamus are essentially sensory, the manifestations of the corpus striatum purely motor. The rôle of the optic thalami is that of a relay station in the sensory system. They make a definite break in the sensory pathway. Sensations coming from the periphery are, so to say, sorted out here. Some continue their way to the cerebral cortex, where they cause voluntary reactions—reflex movements of special exactness. The others pass to the corpus striatum and produce reactions essentially automatic or elementary. The corpus striatum may be considered as an automatic center, entirely independent of the cortex. Its alterations produce a disorder of muscular function, which is characteristic of the various striate syndromes. All the fundamental ele-

ments, muscular rigidity, attitude, loss of associated movements, marked physiognomy, spasmodic movements and athetochoreic movements, are due to disturbance in the coordinating automatic motor system.

The author feels that the foregoing is a fair summary of our present knowledge of the central gray nuclei. A fruitful harvest may be the result of further study in this fertile field.

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STUDIES IN FOCAL INFECTION: ITS PRESENCE AND ELIMINATION IN THE FUNCTIONAL PSYCHOSES. NICHOLAS KOPELOFF and CLARENCE O. CHENEY, *Am. J. Psychiat.* **11**:139 (Oct.) 1922.

THE ETIOLOGY AND TREATMENT OF THE SO-CALLED FUNCTIONAL PSYCHOSES. SUMMARY OF RESULTS BASED UPON THE EXPERIENCE OF FOUR YEARS. HENRY A. COTTON, *Am. J. Psychiat.* **11**:157 (Oct.) 1922.

One could scarcely find a more strikingly effective instance of the "deadly parallel" than is presented by the simultaneous appearance of these two contributions to the theory of focal infections as etiologic agents in the production of "functional" mental disease. It is fitting and logical that the two articles be reviewed together, yet the task is neither a simple nor an enviable one. No amount of careful and unbiased analysis makes it possible to evade the inevitable conclusion that attacking an identical problem with practically the same weapons, the authors have nevertheless arrived at diametrically opposed conclusions. It would seem proper first to summarize the actual work accomplished by each observer, and then to add the comment which seems to be suggested and justified by the investigations themselves and the remarks of those who took part in the discussion of this important topic.

Kopeloff and Cheney sought to solve the problem of the existence or non-existence of a causal relationship between focal infections and psychoses by carrying out a carefully controlled therapeutic experiment. In the control group, totaling thirty-three, there were fifteen instances of dementia praecox, fifteen of manic-depressive disease and three psychoneurotic patients; while the group of twenty-seven in whom sources of infection were attacked by surgical procedures was made up of seventeen schizophrenic, nine manic-depressive and one psychoneurotic patient. Care was exercised in selecting the two sets of patients so that they were well-balanced in respect to age, sex, duration of psychosis, diagnosis, prognosis and infective condition of teeth and tonsils. For instance, from the standpoint of dental pathology, 101 teeth in the "controls" were pronounced septic, while in the "operatives" 139 were extracted. All but two of the latter group had had extractions performed and, in addition, sixteen had had tonsillar enucleations. The two questions which the investigators attempted to answer were: "What do these groups of cases show comparatively with respect to recovery, improvement, or unimprovement?" and "Has the removal of focal infections in teeth and tonsils brought about recovery or improvement in individual cases?" The table which is here reproduced would seem to justify the conclusion of the authors that "On the whole, then, the operated group appears to have received no more benefit than the control group." Before attempting to answer the second query, and indeed before actual estimations as to the extent of the focal infections were undertaken, Cheney recorded a diagnostic-prognostic survey from the standpoint of clinical psychiatry. These opinions were not consulted in pro-

nouncing judgment on the end result, but a subsequent comparison showed "that no recovery has taken place that was not prognosticated before any focal treatment was employed. We have no evidence on which to base a conclusion that the removal of focal infection has of itself brought about recovery."

Since he had not undertaken an experimental study, Cotton's article is justifiably more general in its tone. He has previously written in terms specific enough, and his published results in individual cases are known to every student of psychiatry. Since 1918, his recovery rate in the "functional" group (manic-depressive, dementia praecox, paranoid states, psychoneuroses) has averaged 80 per cent. Complete faith in the specific effectiveness of detoxication treatment (by which is implied the surgical treatment of infected teeth, tonsils, uterus, seminal vesicles, colon, etc., plus the injections of gastric autogenous vaccines and antistreptococcic and anticolon serums) is strongly reiterated. Summaries of stomach analysis in 106 psychotic and fifty-six nonpsychotic persons are presented. The following sentence strikes the keynote of the conclusions: "We have produced evidence, both clinical and pathological which should set at rest any doubt as to the accuracy of our deductions."

Granting that Kopeloff is a competent bacteriologist and that Cotton has reliable laboratory facilities, one is faced with a mass of conflicting technical evidence. For instance, Kopeloff found it a matter of great difficulty to secure

RESULTS WITH AND WITHOUT OPERATION

	Dementia Praecox		Manic-Depressive	
	Controls	Operatives	Controls	Operatives
Number of cases.....	15	17	15	9
Recovered.....	..	..	5	4
Improved.....	5	5	8	1
Total benefited.....	5	5	13	5
Unimproved.....	10	12	2	4
Left hospital.....	3	5	6	3

cultures from uncontaminated teeth. No organisms were isolated from the tonsils which are not present in the mouths of healthy persons. The value of the Rehfuß method of gastric analysis as a demonstrator of foci of infection is seriously and pointedly questioned. Furthermore, he believes that the bacterial content of the stomach is influenced more by the swallowing of saliva than by the degree of acidity. The simultaneous aspiration by three Rehfuß tubes gave varying degrees of acidity. It seems that the use of a dental suction pump to take up the saliva reduced the number of stomach bacteria from 48,000 to 32 per cubic centimeter. Cotton not only makes a categorical denial of scientific verity of these objections, but particularly calls attention to contaminating organisms which he himself did not encounter, and he insists that Kopeloff's series of examinations was entirely too small to be worth while. In only a few of Cotton's patients did the organisms in the stomach correspond to those in the mouth. One of the members in his discussion called attention to Schottmüller's inability to find any infective condition which he could ascribe to oral sepsis in 10,000 bacteriologic examinations (*Deutsch. med. Wchnschr.*, Feb. 9, 1922). Doubtless, expert opinions to the contrary could be as readily quoted. While it is possible that the answer to the much mooted question is to be found in the bacteriologic technic, it may not be decided by the opinion of one or two or even several men, and must be eventually answered by evidence still to be accumulated.

What is the present position respecting the rôle of focal infection in psychiatry? How are we to decide whether we are confronted by a theory or an established fact? Kopeloff's and Cheney's well-balanced and carefully controlled experiment should be given some weight. While it is true that they worked with small groups, they were scarcely small enough to make it possible to attribute the negative result to mere chance. Some investigators feel that they have been able to cure their patients; others complain of failure. Nothing is more difficult than the decision as to just what agent or agents contributed to a recovery in a given case. The issue is heavily clouded by the possibility of diagnostic errors. Cheney's preliminary diagnostic-prognostic survey was a wise precaution, which should be repeated in further studies. A benign psychosis may readily be mistaken for a malignant one and vice-versa, but the margin of error may be decreased by an initial strict rating from the standpoint of clinical psychiatry.

Cotton's reported results are brilliant. For four years the recovery rate has averaged 80 per cent. The obstacles in the way of accurate follow-up investigations are always tremendous. In dealing with large groups and with the limited number of field workers available, it is hardly possible to accumulate a large amount of specific information. The type and extent of recovery is often a question of medical judgment, and many patients are able to live outside a hospital even though they are far from normal. Four years is too short a time after which to pass final judgment. Manic-depressive psychosis is notably recurrent, and the clear intervals may be long or short, while schizophrenia is far from being in every instance a continuous psychosis. There are at times well marked remissions and not altogether infrequently a cessation of the psychosis or at least of its outstanding symptoms, with or without appreciable defect. To sum up: A clear-cut negative experiment, the lack of agreement among competent observers, the possibility of errors in the follow-up work, the insufficient length of time which has elapsed since the detoxication treatment was put into effect on a large known group, and the uncertainty of certain aspects of the bacteriologic technic are in favor of the opinion that focal infections are not yet to be regarded as specific etiologic agents in the production of psychotic states; and it seems neither ultraconservative nor unscientific to believe that the problem is still in its embryonic stage.

If, as seems to be the case, the majority of neuropsychiatrists rightly prefer to regard the effect of focal infections as problematical, it seems pertinent to inquire into the usefulness of the theory. What are its advantages? Is it guilty of any sins of commission or omission? While each one must trust his own judgment, there is apparently a fairly large group of cases in which no question can arise. I refer to the patients in whom there is a pathologic condition demonstrable by recognized and more or less standardized methods. It seems reasonable and even necessary to remove such an unquestionably pathologic condition whenever possible. Here psychiatry owes a debt to Cotton and his followers for the emphasis which they have placed on the factor of focal infections. There is no good reason for neglecting to remove teeth and tonsils which are indisputably diseased. On the other hand, enthusiasm should not be permitted to outpace judgment, even when the consideration is the removal of a single tooth. Further, any operation which is attended by a mortality of 30 per cent. should not be undertaken unless the indications are clear-cut and definite. It is not an easy matter to decide that the colon is infected. To argue that death from colectomy is preferable to terminal dementia

is scarcely admissible. Clinical psychiatry has not yet attained that diagnostic perfection which makes it possible to predict terminal dementia with any degree of surety, at least not until the psychosis has reached a stage in which even colectomy will not avail.

A new theory never strengthens itself by a display of impatience with other theories. Even the study of heredity, which is dismissed as a "fixed quantity in the equation," has served psychiatry usefully in the past and has not yet outlived its usefulness. Whether or not it has an influence on the prognosis of a given case is a question which seems honestly debatable, but that it does have a far-reaching effect on the mental health of the race is not to be denied. The ardent advocates of the importance of heredity should at least be credited with having the same objective as do the believers in the theory of focal infection; namely, the reduction of the total percentage of mental disease.

Likewise, the psychogenic factor may not be dismissed with two short pages. One need not hold a brief for the psychogenetic conception to feel that not infrequently a strong emotional stirring, or perhaps even more often the accumulation of many less dramatic stresses, may have been the factor which conditioned the departure from reality. It seems unfortunate to revive the age-long question of structure and function, and one may readily agree with Cotton that the concept of a disordered mind and a perfectly normal brain is untenable. However, one might want to specify that the brain like other organs is capable of ephemeral as well as permanent changes, of chemical disturbances as well as organic pathologic alterations. Instead of stating that "disordered function must depend upon and be the result of disordered structure," one might prefer, for the present at least, to say that "disordered function is associated with disordered structure." Experimental physiologists have produced at least temporary structural alteration by the stimulus of an artificially induced emotional state.

It is scarcely fair to imply that psychiatrists who have not been able to accept all of Cotton's views are standing pat "on a do-nothing-policy." There is much that is worth while in the theory of focal infections, and its useful application is not being neglected. There are also elements of importance in other theories which are being turned to good account. In the last analysis, it will probably be necessary, valuable and constructive for some time to come to regard psychoses as nonspecific reactions. In this way, the problem may be attacked from every possible angle and the door of investigation kept open to its widest possible extent. One might remark that the slogan "focal infections or nothing" will not gain many adherents for the theory.

It is regrettable that the public has been taken so freely into medical confidence. With due respect for the value of public opinion, it is scarcely fitting that it be the arbiter of a question which involves so many technical considerations. "Discoveries" in medicine and perhaps particularly in psychiatry should not be broadcasted in the lay press until every reasonable doubt has been removed.

Many years before Cotton gathered together the available data and emphasized their importance, many neuropsychiatrists were carefully scrutinizing their patients from the organic point of view and correcting correctable physical defects, and they continue to do so. In spite of extremists, there has been a healthy tendency in psychiatry to take advantage of newer methods either developed in our special field or borrowed from the armamentarium of inter-

nal medicine, and there need not be the slightest concern that this broad approach will ever be abandoned. The question of focal infection is being and will be thoroughly tested along with other conceptions, and will eventually be accorded its proper position in neuropsychiatry.

It is scarcely a valid objection to state that many public institutions do not have proper laboratory facilities. Neither do they have facilities for the practice of psychanalyses or the study of endocrine dyscrasias, but the reason is obvious. Surely it is not because they have conceived a strong prejudice against the study of focal infections or psychanalysis or endocrine disorders. Fortunately, there are enough institutions, both public and private, in which intensive investigations are carried out in all fields, and a sufficient number of intelligent neuropsychiatrists who study their patients thoroughly from every point of view, to make it impossible for the theory of "focal infections" as causative factors of psychoses to fall into the discard through lack of scientific attention.

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OCULOGASTRIC AND OCULOCOLIC REFLEXES. DANIELOPOLU, RADOVICI and CARNIOL, *Ann. de méd.* **11**:143 (Feb.) 1922.

The authors have shown in previous papers that ocular compression produces, in addition to the oculocardiac reflex, a series of reflexes relating to all the abdominal viscera. They have described an oculo-colic and an oculo-vesical reflex. The most advantageous cases for the study of these reflexes are those in which there is a destructive spinal lesion, which induces in the sublesional cord a pronounced automatism. With the automatism of voluntary muscles, they have shown that there is a visceral automatism for all the organs dependent on the motor nerves of the cord below the level of the lesion.

The distention of the bladder produced experimentally by insufflation, produces a contraction of the muscles of the abdominal wall (and of the thigh). A similar visceromotor reflex applies to the descending colon.

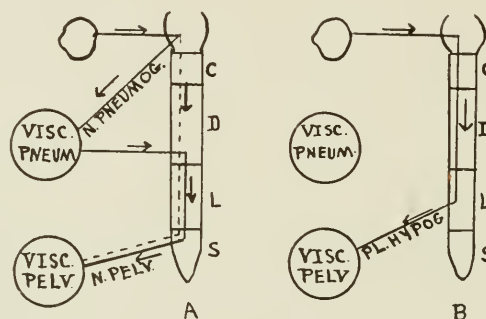
Either pricking or pinching the skin below the level of the lesion gives a cutaneovesical reflex. This excitation of the skin provokes contractions of the bladder wall, accompanied by a desire to urinate. A more intense excitation of the skin, below the lesion, results in both the cutaneovesical reflex and automatic movements of the legs. All excitation of the skin of the trunk superior to the lesion or excitation of the skin of cranial nerve innervation does not cause this reflex.

However, ocular compression, even very slight, brought about an oculo-vesical reflex in the subject with the spinal lesion; the bladder as well as the descending colon contracted with violence, phenomena which they have been able to record by the graphic method. If the compression is strong or prolonged, after the vesical contraction, automatic movement of the legs occur, movements which the subject is incapable of executing voluntarily.

The authors then undertook to demonstrate whether these reflexes are present in the normal person. They used in their experiments a person whom they choose to term amphotonic, because he showed hyperexcitability of both the vagal and sympathetic systems.

The tracings obtained and published with the paper show that ocular compression produced an oculocolic and oculogastric reflex. This reflex generally displayed itself in two phases, a phase of inhibition followed by a phase of exaggeration. The inhibition related both to tonicity and contractility. Likewise, the phase of exaggeration related to both. But for the stomach, it is

especially inhibition that is produced, and the phase of exaggeration which follows is represented chiefly by increase in tonicity and less by augmentation of contractility. They also found that for the stomach the ocular compression produced an inhibition longer in duration directly in proportion as the contractions prior to the compression had been feeble, but that intense ocular compression made the stomach quiet during a very long phase even if, up to that moment, its contractions had been strong. Thus, it appears that ocular compression provoked an enormous slowing of the contracting rhythm, in direct proportion to the intensity of the compression. In brief, the authors agree that the ocular compression has opposite effects in the different organs; an effect predominately sympathetic (inhibitory) on the stomach and an action predominately vagal (inhibitory) on the heart. They further conclude that the oculo-visceral reflexes are conducted by two paths, the sympathetic and parasympathetic (vagal), and that the results obtained depend on the respective



A, possible path of the oculoparasympathetic reflex of the colon; *B*, probable path of the oculosympathetic reflex of the colon.

tonus of the two autogonistic groups of each organ. The reflex is carried by each path to each organ, but predominates on the nerve which presents the higher inherent tonus.

The remainder of the paper deals with a study of the nerve pathways concerned and can be summarized by the diagram presented.

DAVIS, New York.

CLINICO PATHOLOGICAL NOTES ON SOLITARY TUBERCLE OF THE SPINAL CORD. WILLIAM THALHIMER and GEORGE B. HASSIN, Jour. Nerv. & Ment. Dis. 55:161 (March) 1922.

A man, 28 years old, had been complaining of backache and inability to bend his back for about a year. In the course of eight months he developed intermittent sharp and dull pain in the spine between the shoulder blades, with gradual progressing spastic paralysis of the lower extremities associated with a feeling of numbness. There was a sense of numbness in the rectum and bowels and no knowledge of the flow of urine. The reflexes of the lower extremities were increased, more so on the left; the right abdominal reflex was absent. On the right side of the trunk, there was impaired sensation to pain and touch below the seventh rib; on the left side, a band of hyper-

esthesia at the level of the umbilicus was present. Serologic findings were negative. Roentgen-ray examination revealed osteophytes of the entire lumbar and eleventh and twelfth dorsal vertebrae. The spinal fluid on a few occasions revealed a typical Froin syndrome. A diagnosis of spinal cord tumor was made and enucleated at operation, at the level of the second dorsal vertebra. There was no improvement of the patient's condition, and death followed a few weeks later.

Necropsy Findings: These were: tuberculous scars at the apexes of both lungs, bilateral iliopsoas tuberculous abscess and osteo-arthritis of the spine which the authors regard as probably tuberculous in origin, but a careful examination of all the bodies of the vertebra revealed no tuberculous lesions. The tubercle was made up of numerous miliary tubercles with fibrous formation and cheesy degeneration. All ectodermic tissue was displaced by the tubercle. There was typical secondary degeneration of the spinal cord above and below the tuberculous mass. There was extensive leptomeningitis, pachymeningitis, and peripachymeningitis with involvement of the spinal roots, especially marked at the level of the spinal cord harboring the tubercle. The subdural and sub-arachnoidal spaces were entirely obliterated. There was total absence of inflammatory phenomena of myelitis in the areas not affected by the tuberculous process.

The authors' conclusions drawn from the pathologic study of this case are: that a solitary tubercle of the spinal cord is the outcome of a local inflammation of the tissues brought on by a specific micro-organism; that the inflammation provokes reactive phenomena on the part of the surrounding tissue elements causing proliferation of the adventitial cells and of hematogenous elements (lymphocytes, plasma cells, etc.); that the adventitial cells, filled with the detritus taken from the inflammatory area, are drained into the subarachnoid space where they provoke various inflammatory reactive phenomena on the part of the pia-arachnoid, that is, a meningitis. It is the authors' claim that the latter is a secondary process, and not due to direct invasion of the meninges by the tubercle in the spinal cord; likewise, that the draining of the disintegrated material by the subarachnoid space as well as the powerful connective tissue rings around the tubercle are responsible for the preservation of the rest of the spinal cord and the absence of myelitis.

The authors have been able to collect sixty-seven reported cases from the literature, which appeared to be definitely proved cases of solitary tubercle of the cord, fifty-eight of which are abstracted in their article. In reviewing these, it has been noted that many of the tubercles were multiple and some situated in the brain and brain-stem as well as the cord. Inflammation of the meninges was associated with it in the majority of cases.

STACK, Philadelphia.

NEO-ARSPHENAMIN IN HYPODERMIC INJECTIONS. MINET and
LEGRAND, *Rev. de. med.* **39**:230 (April) 1922.

The authors recall that the first injections of neo-arsphenamin were made into the muscles. In acid or alkalinized solution, it was injected in the supero-external portion of the buttock, above the great trochanter near the iliac crest. Such injections were excessively painful and produced great lesions of infiltration or necrosis. To obviate this, oily mediums were tried, but because the absorption of such mediums was poor, they did not become a vehicle of choice. When they were abandoned the method of intravenous injection rapidly

increased. The discovery of neo-arsphenamin in 1912, by Ehrlich, simplified the matter as did Ravaut's demonstration that even the largest dose of neo-arsphenamin after being dissolved in the small amount of distilled water which the ampule itself can hold, can with safety be injected intravenously with a small hypodermic syringe and needle.

In spite of this consideration and the increasing inclination to use the veins for medicinal injections, the authors do not find themselves able to ignore the several disadvantages which they feel are inherent in the intravenous method. One disadvantage is the serious trouble that one sometimes has in finding a difficult vein, and the disturbing effects if even a small portion of the solution is allowed to flow outside the vein. Another disadvantage is the accidents, immediate or late, which may result. Mentberger, in 1914, published a statistical report of 229 cases of death from intravenous injections.

More frequent are the so-called nitritoid crises of Milian, a vasomotor syndrome accompanied by a syncopal or lipothymic state. Other accidents assume a gastro-intestinal form—violet gastralgia, sudden diarrhea or vomiting. Sometimes fever, chills, cephalalgia and a so-called Herxheimer reaction develop. Later one observes albuminuria, cutaneous eruptions and late nervous phenomena.

In spite of the widespread use of the intravenous method, therefore, the authors have not rallied to it; instead they early became the protagonists of the method of subcutaneous injection of neo-arsphenamin which Sicard, in 1920, thoroughly reviewed. They believe that by this method they can assuredly avoid nitritoid crises and all the other occasional late and early disagreeable reactions. Further, they are convinced that they can realize a more intensive treatment than is possible with intravenous injections. Sicard never goes beyond 0.15 gm. for an injection. Trossarello does not hesitate to give 0.75 gm. The authors use from 0.05 to 0.30 gm. In one case, desiring a very intense action, they injected 0.45 gm. daily, without any bad effect.

The technic followed is that described by Wechselmann and used also by Sicard. The place chosen for the insertion of the needle is the external surface of the thigh, principally the trochanteric region. The neo-arsphenamin is dissolved in 0.5 to 1 c.c. of dissolvent. It is necessary to push the needle so that the injection is made immediately superficial to the fascia lata. To establish this, one needs to find the level at which the needle can be readily displaced in all directions without catching in the trabeculae of the subjacent aponeurosis. Finally, it is necessary to perform the procedure rapidly.

After experimenting with various dissolvents, they found that a glucose solution—47 parts to 1,000—highly satisfactory, but more recently they have used with even better results the glucose solution with phenic acid, the exact proportions of which are not stated. In their paper, the authors also make clear that in conjunction with these injections, they simultaneously subject the patient to mercurial and iodid treatment.

DAVIS, New York.

A TRIAL OF THORIUM X IN PSYCHIATRIC THERAPEUTICS.
P. DADAY, R. BESSIÈRE and L. JALOUSTRE, *Presse méd.* **30**:520 (June 17) 1922.

This substance loses half its strength about every three and one-half days, and is practically inactive after twenty days. One result of this property is the absence of cumulative effects. The preparation used in the experiments was an isotonic solution of bromid of thorium X, put up in ampules contain-

ing a variable quantity of the active substance, from 10 to 1,000 micrograms in strength. Injections could be given subcutaneously (the method of choice), intramuscularly or intravenously. The drug might also be administered by mouth, if not over 150 micrograms were given in a day.

Each patient was given a series of five injections, one week apart, then after four weeks' rest five more injections were given spaced as before. No dose consisted of less than 350 or more than 700 micrograms.

Local pain was occasionally complained of for several hours after an injection, and was accompanied by a transitory elevation of temperature. Nausea and faintness were sometimes evident. During the second course of injections, almost all the patients exhibited a local or a more or less general pigmentation of the skin, which disappeared in about a fortnight after the last dose. There was also a concurrent tendency to slight anemia and loss of weight. Two patients developed mild ulceration of a finger.

Three groups of patients were treated, diagnosed as having (1) involutional melancholia, (2) dementia praecox, and (3) acute confusion. No improvement was observed in the first group (nine cases). In the second group, the two patients with paranoid praecox and the four of simple praecox of long standing were unimproved, while one with recent simple praecox was apparently cured. The third group comprised three patients, two of which were cured and one improved.

The authors are confident that their confused patients recovered far more rapidly with this treatment than they would have done without it. It was shown that if improvement was to take place in a given case, it would be well marked after the second or third injection. The value of a second series of injections remains doubtful. No theories are offered on the mechanism of the cure.

HUDDLESON, New York.

THE PROCESS OF INGESTION IN THE CILIATE, *FRONTONIA*.

WILLIAM M. GOLDSMITH, J. Exper. Zool. **36**:333, 1922.

Observations and experiments were made on the ciliate, *Frontonia leucas*, while the organisms were ingesting euglenas, diatoms, desmids and oscillatoria filaments. The ingestion of blue-green algae, especially oscillatoria filaments, furnished the most conclusive demonstrations of the method involved, as the process continued a greater length of time and involved more factors than did the ingestion of smaller organisms. Five factors were involved in the process of ingestion of material longer than the expanded width of the body of *Frontonia*. In case of smaller particles, the third, fourth and fifth factors mentioned below are not essential to ingestion.

(a) Action of Oral Cilia: The cilia about the mouth of the frontonia exert a direct pull on the incoming food.

(b) Action of Locomotor Cilia: The cilia of the body in general drive the organism forward and thus force the stationary food into the mouth.

(c) Rotation of Body Axis: The end of the fiber usually enters the mouth and passes anterodorsally until it comes in contact with and exerts a pressure on the aboral wall, after which the frontonia swings around through an angle of almost 180 degrees, using the mouth as a pivot. This change of position permits the fiber to pass dorsally along the aboral side of the ciliate. Through the play of either factor *a* or *b*, or both, ingestion continues until the fiber exerts such pressure on the body wall at the extreme posterior end that the organism is extremely elongated and pointed. Such pressure on the body wall

acts as a stimulus, causing movements that relieve the stimulation. The rotation of the body axis assists in relieving the stimulation at certain of these tension points.

(d) Body Contractions: A series of sharp contractions of the body wall assists in relieving certain other tension points.

(e) Cyclosis: Cyclosis aids by moving the end of the fiber around the wall, thus making further ingestion possible.

Unusual and fantastic figures are produced through the contortion of the organism by the ingested food which varies in size, shape, density, elasticity and color.

WYMAN, Boston.

AN EXPERIMENTAL STUDY OF THE MECHANISM OF HALLUCINATIONS. MORTON PRINCE, J. Psychol., Med. Sect. 2:165, 1922.

Postulating that artificial hallucinations or "crystal visions" are identical with many of the hallucinations of the insane as well as of the sane, Prince here records a series of observations made on a subject "who both experiences hallucinations and can produce automatic (subconscious) script without awareness of what the hand is writing." In a first set of studies the subject wrote automatically regarding some topic suggested to her and at the same time indicated the moment of appearance and disappearance of any "hallucinations" which occurred during the writing. These hallucinations were described from memory, either at the time or subsequently. In a second series, hallucinations were induced by crystal gazing, this time without any suggestion as to topic, and while describing the visions the subject performed automatic writing. In both series "the method of subconscious introspection," answering questionnaires in automatic script, was employed to obtain details of the content of the subconscious during the time of the experiments.

The results show clearly that the conscious images (hallucinations) were correlated with, and followed the train of, subconscious thought. Furthermore, it was learned that an imagery occurred in the subconscious similar to that which accompanies conscious thought, though without awareness, and that this preceded in time the conscious hallucinations. In the second series of experiments, the script showed some striking misspellings and distortions, besides being so brief and symbolic as to show at first glance little connection with the vision. This was explained by the fact that the subject of the experiment had previously suffered from dissociation into a "dual personality." In the automatic writing, the full statement of the happenings in the subconscious was secured only by "tapping" the second personality system.

The principal conclusions reached from this series of careful records are that such dreamlike fantasies whenever encountered are "indications of a dissociated subconscious process and its content." "The genesis and psychopathology of the psychosis are to be found in the forces which have determined the dissociation and motivated the subconscious process."

SINGER, Chicago.

THE SIGNIFICANCE OF NYSTAGMUS IN A LESION OF THE ABDUCENS NUCLEUS. JOSHUA ROSETT, Neurol. Bull. 3:332 (Sept.-Oct.) 1921.

On June 1, 1921, the patient, aged 52, awoke in the morning to find that his face had been drawn to the right side and that he could not close his left eye. He had double vision, which became worse in the course of the day, and which has persisted to date.

Except that he was a heavy drinker for a number of years, his history was negative. The interesting physical findings were: paralysis of the external rectus muscle of the left eye; inability to move the right eyeball to the left when a similar effort was made on the part of the left eye; rhythmical nystagmus of 1 mm. at the rate of four a second when the eyes were turned in a horizontal direction to the right; flaccid paralysis of the facial muscles of the left side; and definite loss of gustatory sense on the anterior two thirds of the left side of the tongue. About ten weeks later all these symptoms disappeared, with the exception of paralysis of the external rectus muscle of the left eye.

The author localizes the lesion around the sixth nerve nucleus, which, extending peripherally, would catch the seventh nerve fibers as they hook over the sixth. The *nervus intermedius* carrying the fibers of taste would be caught by the lesion extending a few millimeters out from the abducens nucleus as the former bends down to enter the nucleus solitarius. The fact that a complete lesion of any nucleus must cut off the pathways connecting it with all other structures accounts for the inability to move the right eye to the left in the effort of conjugate movement of both eyes, while the right eye can be turned inward in a movement of convergence. The author's theory in regard to nystagmus is that there is no connection between the vestibular nuclei and the nuclei of the oculogyric mechanism, as in the case of all the other motor nuclei of the central nervous system. The explanation of this is based on the fact that the center of gravity is not changed in the eyeballs on movement of the body, and that the adjusting of the visual axes to the body and head would be entirely between the optic mechanism and the cerebellum. Assuming this line of reasoning to be correct and the nystagmus in the case of the patient mentioned a mode of incoordination of the nature of dysmetria, the author assumes that there is an injured conduction pathway from the cerebellum to the nucleus of the sixth nerve, an interrupted rubro-abducental tract.

In the discussion of the author's case which followed, the opinion was given that the nystagmus was neither vestibular nor cerebellar, but a paretic ocular nystagmus due simply to imbalance in the muscles, entirely peripheral in nature.

STACK, Philadelphia.

AN EXPIATION PROCESS IN A CASE OF SCHIZOPHRENIA. HENRY DEVINE, *J. Neurol. & Psychopath.* 2:224, 1921.

The report is of exceptional interest for the reason that the account was given spontaneously by the patient, without suggestion by the examiner. The analysis has been limited strictly to the facts and is admirably clear and connected. The chief features in the outward appearance of the patient were a wandering, shut-in existence extending over many years, stilted phraseology with many mannerisms and neologisms, an attitude of mocking humility and exaggerated politeness and episodes of extreme irritability and antagonism.

When secured, the detailed story revealed the presence of hallucinations, called by the patient "strengths," which referred to offenses actually committed against parents, friends and others during childhood. The mannerisms and attitudes of the patient were clearly stated to be expiatory penances which he was compelled to repeat innumerable times for these offenses and for any similar thought which he might entertain even for a moment of similar activities now. The "strengths" would trick him into such thoughts, often obscurely symbolic, in order to have the pleasure of punishing him.

Dr. Devine concludes that these indicate an ambivalent tendency toward sadism and masochism, using these terms, not in relation to specific sex urge of which the patient spoke hardly at all, but rather in the sense of a "more fundamental than even sex" instinct which he suggests corresponds with Schopenhauer's "will to live" or Nietzsche's "will to power." The richness and fulness of intellect revealed by the patient in his descriptions are stressed as evidence against any true dementia, and the conclusion is reached that "the psychosis is the outcome of an arrest or fixation of the instinctive and emotional life at a primitive or infantile level," in spite of the high level of intelligence which has been reached.

SINGER, Chicago.

PAIN IN THREATENED AND REAL GANGRENE OF THE EXTREMITIES: ITS RELIEF. BERNHEIM, *Am. J. M. Sc.* **163**:517 (April) 1922.

The author does not attempt to suggest a cause for the intense pain in gangrene of the extremities. He says that he is at a loss to explain it. Pain, however, is an early and constant symptom of the disease, and first manifests itself as a numbness and tingling of the toes and feet, together with a sensation of cold and a marked blanching of the extremities. The patient usually first complains of pain in the extremity so severe that it wakens him at night. The condition is often mistaken for a neuritis, whereas the circulatory system is at fault. There is no nerve degeneration. Relieving the gangrene relieves the pain, and measures are instituted for this purpose.

The pain is usually so intense that huge doses of sedatives are required, the most efficient being sodium bromid in cautious doses. Rest is important. Blood vessel exercise is advocated by plunging the leg first in a pail of ice water and then after a minute into a pail of very hot water for a few seconds. The process is kept up for five minutes, and finally a prolonged bath in the hot water brings vascular dilatation. This is repeated three times a day if the patient can stand the procedure. Following the hot and cold plunges, the member is dried and rubbed with olive oil. An electric vibrator is then used similar to that used on the face in barber shops. This is applied for ten minutes at a time for several applications daily.

One liter of Ringer's solution is given into the duodenum by tube each day until the patient becomes unable to take the treatment.

The Moskowitz method of testing the reaction time of the vessels is used once a day. The member is kept clean and all precautions taken to prevent ulceration. Strict palliative measures are suggested. Amputation is to be the last resort. Arteriovenous anastomosis has not proved satisfactory. The Leriche operation for stripping the vessels has not been tried by the author; he feels that it requires more confirmation.

When operation is resorted to, the limb should be amputated high enough to ensure enough blood for healing of the flap. This the author has found occurs only when the member is taken off above the knee. Amputations at lower levels have to be repeated.

TEMPLE FAY, Philadelphia.

A STUDY OF THE PHYSIOLOGY OF THE NERVOUS SYSTEM OF INSECTS. II. RESEARCH ON CIRCUS MOVEMENTS PROVOKED IN COLEOPTERA. EDGARDO BALDI, *J. Exper. Zool.* **36**:211, 1922.

Injury to one side of the supra-esophageal ganglion of certain beetles brings about a change in behavior which is in harmony with the chiasmatic

arrangement of the fibers running from the brain to the lower ganglions, not being restricted to one lateral half of the body but involving the whole organism. This is especially clear in the antimeres opposite to the lesion. This injury probably provokes a general increase of movement which is manifest in the appendages of the injured side. The final effect is a disturbance of equilibrium in the normal functioning of the musculature, in which the activity of the flexor muscles of the uninjured side predominates. Thus movement in a circle is produced. The movement in a circle is due for the most part to the traction predominatingly exercised by the first two appendages of the uninjured side, this traction being variously directed with respect to the sagittal plane. It is helped by the propulsive arcuate movements characteristic of all the appendages of the injured side. That the movements of flexion are the principle cause of circus movements is proved:

(a) By the impossibility of eliciting such movements by the artificial exaggeration of the propulsive activity of one side.

(b) By the reestablishment of straight locomotion, when, by amputation of leg segments, the movements of traction are rendered impossible.

(c) By the persistence of circus movements where only the propulsive arcuate movements are prevented in the legs of the injured side. Thus circus movement results from an alteration of the general conditions of symmetry of the organism, but from such an alteration as affects the entire organism, assuming various aspects in different regions.

WYMAN, Boston.

A CASE OF VENOUS ANGIOMA OF THE CEREBRAL CORTEX.

H. CAMPBELL and C. BALLANCE, *Lancet* 1:10 (Jan. 7) 1922.

These authors report the case of a man, aged 23 years, who at 13 first noticed weakness in the left arm and leg, which increased progressively. At about the same age, he developed seizures which began with numbness in the left great toe and extended upward. When the numbness reached the left hand, it closed; and when the left temporal region was reached, the head was drawn to the left and the mouth opened. These seizures were followed by severe headaches. Until three months prior to admission (Nov. 1, 1920) the seizures occurred every six months; but they became more frequent and were accompanied by clonic spasms. Examination on admission showed the presence of a mild degree of left hemiplegia with increased deep tendon reflexes on that side, and loss of the left abdominal reflex. Ankle clonus was present on the left side, but no Babinski's sign could be elicited. There was diminished sensory discrimination over the left foot, leg, arm and forearm. The optic disks were normal. A diagnosis of a progressive neoplasm involving the right precentral and post-central convolutions was made. At operation, April 2 and 9, 1921, an extensive venous angioma covering the external and mesial surface of the right cerebral hemisphere was found. The nevus grew from the vessels of the pia and cortex and could not be removed. Six months after operation, he had had only two convulsive seizures. Campbell and Ballance believe that the long history (ten years), the slowly progressive left-sided paresis, the absence of optic neuritis, seizures commencing with a sensory aura of the left great toe, failure to localize light touch on the skin of the left foot, leg, hand and forearm, point to the presence of a widespread chronic cortical lesion.

POTTER, Mercer, Pa.

A CASE OF PROGRESSIVE MUSCULAR DYSTROPHY. E. D. FRIEDMAN,
J. Nerv. & Ment. Dis. **54**:294 (Oct.) 1921.

The patient, a schoolboy of 16 years, complained of weakness of both legs for fifteen years. His family history was negative. He began to walk at 9 months of age and walked for one month. His legs then became weak, and he crawled until he was 5 years old. After that he was able to stand again, but he noticed weakness of his legs which has persisted. Physical examination showed an attitude and gait characteristic of muscular dystrophy. The patient had a waddling gait and a marked lordosis and arose from the floor with difficulty. The muscles involved were: the sternomastoid on the left, the right spinati, the deltoids, the left pectorals, the left biceps, and the erector spinae. Both quadriceps muscles were weak, with little muscle tissue remaining. The adductors of the thigh were involved as were also the dorsiflexors of the feet. Both knee reflexes were absent. Disturbance in the glands of internal secretion was shown by an overgrowth of hair at the bridge of the nose, the presence of large hands and feet, and female distribution of pubic hair. All laboratory reports were negative. There was no response to galvanism or faradism in the quadriceps muscles.

That the case belongs in the group of muscular dystrophies is evidenced by the combination of atrophy and hypertrophy and the absence of both fibrillation and reaction of degeneration; likewise, to some extent by involvement of the proximal group. The author believes that the lesion is at the neuromuscular junction, because there is no definite symmetry in the localization of the lesion. He thinks this might explain the absence of knee jerks in this case. He also believes that all cases in which no pathologic condition can be demonstrated in the anterior horn cells or their axons should be grouped under the term muscular dystrophy.

The pathologic picture of this disease is usually that of a primary muscular atrophy. There is atrophy and hypertrophy of the muscle bundles with increase in the number of nuclei, proliferation of the perimysium, a deposit of fat cells in the muscles and vacuolization of the muscle cells. Erb looks on hypertrophy as a preliminary stage of the disease.

The prognosis is good unless the muscles of respiration are involved. The disease may last from thirty to forty years.

STACK, Philadelphia.

CONCERNING DIFFUSE GLIA REACTIONS. J. L. PINES, Schweiz.
Arch. Neurol. u. Psychiat. **11**:112, 1922.

A number of authors, including Henneberg, believe that the normal glia cell may be transformed into a glia tumor cell and that no sharp division is possible between a diffuse glioma and a secondary reactive glia proliferation; also that the proliferation does not start from a focus of glia cells, but is diffuse from the outset. Ceni and Chiari share this belief. Stumpf believes that tumor cells of this type proliferate in the pre-existing neuroglial syncytium, the irritant bringing about a progressive proliferation of glia cells. Similar conclusions were drawn by Landau, who assumes a rather general congenital abnormality. The author believes that the increase in glia cells in his case is reactive and gliomatous in character.

As irritants, Merzbacher includes tumor, hemorrhage and softening; however, these factors do not seem sufficient in themselves to result in true glioma formation. According to most authors, these determinants reside in the glia

cells themselves, which, according to Cohnheim, as is well known, represent displaced embryonic cells. Lenhossek suggests agenetic, or incompletely developed glia cells which have not been implanted from the outside, but which retain their embryonic character and fail to differentiate fully. Exactly what is required for the production of diffuse reactive glial proliferation approaching the tumor type of cell, in contradistinction to the normal glial cell reaction, is not yet known. The organs of internal secretion, such as the choroid plexus, have been looked on as supplying the required stimulant. Several authors have laid great stress on the association of glioma with other constitutional anomalies. Anton called attention to a case showing diseased suprarenals and hypertrophy of the brain and persisting thymus. Neumann reported a case of glioma, cyst of the pineal body, persistent thymus and enlarged thyroid. Bartel reported a series of gliomas associated with status thymicolymphaticus. Pines believes that this is more than coincidence.

In conclusion, he believes that there was present in his case an individual factor expressing itself in heightened glia reaction, the precipitating moment being furnished by the irritation resulting from the primary dural tumor.

WOLTMAN, Rochester, Minn.

CASE OF SPINAL SPASTIC PARALYSIS (PRIMARY LATERAL SCLEROSIS). J. T. BROADWIN, *J. Nerv. & Ment. Dis.* **54**:289 (Oct.) 1921.

A man, 29 years of age, complained of stiffness in both lower extremities, which began at about 12 or 13 years of age, progressed rather slowly up to three years ago, and has remained stationary since then. The family history was negative. At 16 the patient noticed that he wore out the inner sides of the soles of his shoes rapidly. He was unable to state which leg was first involved. There were no subjective sensory symptoms other than occasional transient pains in his feet.

The interesting physical findings were: coarse nystagmoid movements with a rotary element in both eyes on fixation to extreme lateral planes; a fine tremor of the fingers of the outstretched hands; spastic gait, the right leg appearing more spastic; the legs moving en masse as though walking on stilts, with a scraping of the feet. Considerable resistance was encountered during passive movements of both lower extremities, more marked on the right. The gross motor power was well preserved. There were no atrophies or fibrillary tremors, and likewise no objective sensory findings. There was a slight lordosis in the lumbar region, a roentgenogram of which revealed no abnormal findings. The reflexes of the upper extremities were lively; the knee and ankle reflexes were exaggerated; patellar and ankle clonus and a positive Babinski sign were present.

Oppenheim states that in the great majority of cases that first appear to be a spastic spinal paralysis, symptoms later appear which show that behind this picture there frequently lurks some other disease of the nervous system, such as disseminated sclerosis, chronic myelitis, compression of the cord and occasionally amyotrophic lateral sclerosis or cerebral disease. Necropsy findings of this disease have formerly been reported by Erb, Spiller and Strümpell. The course of the disease is slow, and it may even cease to progress.

In the treatment of severe forms, division of certain of the posterior lumbar roots is sometimes advised.

STACK, Philadelphia.

AN ANGIOMA OF THE PONS. LEYSER, Monatsch. f. Psychiat. u. Neurol. **51:83** (Feb.) 1922.

Angioma of the pons is a rare condition. Only seven cases are reported in the literature. The author's patient was a woman, aged 20, always well up to the present illness. The onset of the illness was sudden. She cried out, fell to the ground, and had a convulsive seizure. After half an hour, there was deep unconsciousness. The pulse was slow, respiration irregular; there was vomiting, and incontinence of urine. The pupils were small, equal and rigid. The left corneal reflex was gone. Jerking movements of the left arm and leg and flaccid paralysis of the right arm and leg were present. On the fourth day, there was a beginning left facial paralysis, which increased to complete paralysis in a few days. There was a skew deviation of the eyes, the left eye pointing down and in, the right eye up and out. With the exception of a slight clearing on the tenth day, the patient remained deeply unconscious to the end. Death occurred on the twentieth day.

Necropsy revealed a small left-sided angioma of the pons, with an extensive hemorrhage which had caused much tissue destruction, and had broken into the ventricle.

The most striking feature was the so-called Hertwig-Magendi symptom, or skew deviation of the eyes. The eye on the affected side turned down and in, the eye on the opposite side, up and out. The pathologic basis for this is not definitely known. Some consider it due to a lesion of the middle cerebellar peduncle. Biehl believes it to be due to an affection of the vestibular nucleus. The author attributes the symptom in his case to the destruction of the left vestibular nucleus.

The combination of this left-sided skew deviation with a right-sided hemiplegia forms a type of hemiplegia alternans hitherto undescribed in the literature. The author suggests as a name "hemiplegia alternans vestibularis." The syndrome will probably never be found pure, but associated with involvement of the homolateral abducens and facial nerves.

SELLING, Portland, Ore.

RECURRENT HYPERTROPHIC NEURITIS. F. J. NATTRASS, J. Neurol. & Psychopath. **2:159**, 1921.

A remarkable case, apparently unique in the literature, is grouped by the author with the "hypertrophic interstitial neuritis" of Déjerine and Sottas in spite of marked differences, notably the recurrences. The patient had suffered two apparently similar attacks, which had been regarded as anterior poliomyelitis. The first occurred at the age of 4, with recovery in six months, and the second at the age of 17, with recovery in three months. The third attack began suddenly at the age of 18, was afebrile and progressed rapidly to almost complete paralysis, accompanied by pains, numbness and tingling, in two weeks before the patient came under observation. He then presented flaccid paralysis of the arms, most marked in the distal segments, with atrophy and electrical changes which in the hands constituted a complete reaction of degeneration. The reflexes were absent, and there was tremor on movement with well marked adiadokokinesis. The trunk muscles were weak, but the abdominal reflexes were present.

In the lower extremities, only slight movements were possible at the hip and knee; below, paralysis was complete with atrophy and reaction of degeneration. The reflexes were absent.

The cranial nerves were not involved, and there was no loss of sphincter control. Objective sensory disturbances, with the possible exception of defective pallesthesia, were absent. The muscles were tender everywhere, even in the cranial nerve group. The large nerve trunks (not the cutaneous nerves), where palpable, were uniformly swollen, hard and tender.

Improvement began within a day or two after admission to the hospital, and at the end of three months functional recovery was practically complete, although knee and ankle jerks were still absent, and the electrical reactions were not normal.

SINGER, Chicago.

EPILEPTOID OR FAINTING ATTACKS IN HYPOPITUITARISM.

L. PIERCE CLARK, *Am. J. M. Sc.* **163**:211 (Feb.) 1922.

The occurrence of fainting attacks and epileptoid states in hypopituitarism are considered, and an attempt made to show the relationship between them, clinically and therapeutically.

The seizures usually occur in rapidly growing adolescents who are subject to relatively benign fainting attacks, which may at first simulate larval forms of petit mal epilepsy. They are to be differentiated from the latter condition by the absence of the epileptic character and the general physical and mental stigma of the disorder. The syncopal states are only a part of the obscure clinical picture of dyspituitarism, in which the author states there appears to be an excessive functioning of the anterior lobes of the pituitary gland.

Coincident with the fainting attacks there may be low blood pressure, slow pulse, vasomotor ataxia and a host of muscular and skeletal defects in development. The cause is suggested in the irregular structural growth of the body, resulting in a disharmony of function of the vasomotor and sympathetic systems. It is also pointed out that in the physical sphere one may encounter not infrequently character delinquencies and slow mental development.

The line of corrective treatment advised is outdoor life, exercise and an abundance of sleep plus the administration of specific glandular substance. The author says that patients recover spontaneously after rest and leading an outdoor life. Three cases are cited—in one posterior pituitary and thyroid medication was prescribed, in the other two, more extensive rest. All patients recovered normal balance. The use of glandular therapy is advised in a rational and cautious manner.

TEMPLE FAY, Philadelphia.

ON THE PRODUCTION OF NEURO-MUSCULAR PATTERNS BY THE RELEASE OF SPINAL INTEGRATIONS AFTER DECEREBRATION. W. M. KRAUS and A. M. RABINER, *J. Neurol. & Psychopath.* **3**:209, 1922.

Developmentally, the muscles and nerves may be divided into dorsal and ventral groups. Analysis of the postures of decerebrate rigidity and of the flexion stepping and arm reflex according to the particular groups of muscles involved reveals the following: In decerebrate rigidity the neuromuscular apparatus in action is dorsal for the body axis (trunk and neck), ventral at the shoulder or hip, dorsal at the elbow or knee and ventral at the wrist or ankle and parts below. The neuromuscular pattern for axis and appendages can therefore be expressed by the formula DVDV. In the flexion reflex posture exactly the reverse arrangement is found, VDVD.

A series of cases, three of lethargic encephalitis and one each of a septic meningo-encephalo-myelitis and extradural spinal tumor, are studied and analyzed in this manner, and the essential similarity demonstrated. The encephalitic cases were all of the choreic and kinetic type, and in two of them besides the DVDV action which was common to all three, there were occasional movements of the VDVD pattern. In the two remaining cases, the pattern was fragmentary but true to form.

In a further discussion, it is concluded that the integrations for these patterns must be spinal and that they may be activated by two forces, one kinetic and the other static or tonic, but that the muscles are activated in clearly defined patterns.

SINGER, Chicago.

CALCIFICATION OF THE PITUITARY WITH HYPOPITUITARISM AND WITH SYMPTOMATIC TREATMENT. PFAHLER and PITFIELD, *Am. J. Med. Sc.* **163**:491 (April) 1922.

The roentgen-ray findings in nine cases are presented, with the results of treatment and the use of glandular therapy.

There were varying degrees of calcareous deposit in and about the pituitary region, and the clinical pictures of the cases were surprisingly similar and sufficiently clear to draw attention to the dysfunction of the gland. Fatigue, depression, vagotonia, headache, lethargy, high sugar tolerance, low blood pressure, vasomotor instability, rhinorrhea, belching of gas and absence of nail crescents were the symptoms most often noted.

The article is well illustrated with plates of skilfully made roentgenograms. Four of the cases were found between the ages of 19 and 27, the remaining five between the ages of 38 and 54. In all but one of the cases there were definite symptoms that pointed to pituitary disorder. In the one exception, while there was no definite evidence of pituitary disease, the vagotonia and neurasthenia improved under pituitary feeding. The roentgen ray showed a thickening of the capsule surrounding the gland rather than an infiltration of salts into the substance itself. The authors feel that the presence of such infiltration within the gland is a definite indication of disease either present or past, though in the other cases noted the gland seemed more dense than normally, without any signs of glandular disturbance.

The treatment instituted was the administration of whole gland pituitary in doses as high as 6 gm. a day. Many patients apparently were markedly improved by this treatment. The authors advise the use of stereoscopic plates when investigating disorders of the pituitary.

TEMPLE FAY, Philadelphia.

STUDIES IN ASYMPTOMATIC NEUROSYPHILIS. IV. THE APPARENT ROLE OF IMMUNITY IN THE GENESIS OF NEUROSYPHILIS. ALBERT KEIDEL, *J. A. M. A.* **79**:874 (Sept. 9) 1922.

Investigations show that neural invasion occurs in probably all cases of early syphilis, but clinically less than 50 per cent. of these patients are neurosyphilitic. In the group studied by the author, he found that 21 per cent. of all patients with late syphilis, including cases of latent syphilis, present definite evidence of clinical neurosyphilis; 21.6 per cent. of patients with late syphilis with apparently normal nervous systems have definite abnormalities in their cerebrospinal fluid showing late asymptomatic neurosyphilis, making a total of 42.6 per cent. of all patients with late acquired syphilis who are

actively neurosyphilitic. This represents the percentage of patients with late syphilis who failed to acquire an immunity sufficient to suppress the neurologic invasion. An additional 17.7 per cent. of all those with late syphilis show suggestive evidence of immune protection. The remaining 39.7 per cent. are patients with apparently normal cerebrospinal fluids, in whom neurolatency has developed without demonstrable neurologic damage, or who had developed protection before damage was done. The author concludes that the character of the early tissue reaction to the syphilitic infection, the intensity, continuity and duration of the therapeutic attack, and the occurrence of pregnancy in women, markedly modify the incidence of neurosyphilis, both clinical and asymptomatic.

NIXON, San Francisco.

ON FROIN'S SYNDROME AND ITS RELATION TO ALLIED CONDITIONS IN THE CEREBROSPINAL FLUID. J. G. GREENFIELD, *J. Neurol. & Psychopath.* **2**:105, 1921.

After a brief review of the literature, three cases presenting the complete syndrome and eighteen a partial reaction are outlined. Under this last category are included fluids with a high albuminous content but in which the yellow color may not be marked, or in which there is no spontaneous clotting owing to the absence of fibrin ferment. Addition of the latter will cause clotting. The complete reaction was observed in Paget's disease of the spine, carcinoma of the spine and intradural myxoma. The partial syndrome occurred in cases of acute myelitis, staphylococcus meningitis, cerebrospinal syphilis, thoracic aneurysm pressing on the cord, Pott's disease and spinal tumor. Froin's syndrome is also recorded in the literature as occurring in polyneuritis and Landry's disease.

The author reviews and discusses the theories which have been suggested to explain the condition; and, after pointing out that the fluid approximates in character toward that of the blood serum, concludes that this is brought about by blocking off the fluid in the lumbar sac from that in the spinal and cranial cavities above. The degree of approximation to serum depends on the completeness of the block, and its occurrence is aided by two factors: venous congestion or inflammation with resultant transudation and the entrance of lymph into the subarachnoid space by way of the perineural or perivascular lymphatics. The latter factor explains the occurrence of the syndrome in polyneuritis.

SINGER, Chicago.

THE VALUE OF THE ROUTINE USE OF THE COLLOIDAL GOLD REACTION IN ACUTE EPIDEMIC POLIOMYELITIS. REGAN and CHENEY, *Am. J. Dis. Child.* **23**:107 (Feb.) 1922.

The authors found a distinct diagnostic value in the use of the colloidal gold reaction in their series of twenty-one cases. The curve appeared in all cases, and fell in a definite portion of the reaction zone.

The curve shows a slight rise in the early syphilitic area. It is expressed numerically as 1 1 2 2 1 0 0 0 0 0. The curve lies in the same zone at all periods of the disease. It is highest at the onset of symptoms and therefore has a diagnostic value. There is a gradual fall until the eighth week, but it may persist in cases in which there is a residual paralysis or polyneuritis. A chronic case with recurrent attacks showed a typical curve for five years after the initial onset.

As the curve is similar to a weak tabetic reaction, the history, age and Wassermann reaction must rule out syphilis. Meningismus gives no reaction, so that the use of this test would be of value in differentiating poliomyelitis from that condition. Epidemic encephalitis gives such variable curves that it is easily distinguished from those falling in characteristic zones. In cases in which the curve is prolonged into the higher dilutions (known as the meningitic zone), tuberculous meningitis must be ruled out, otherwise the curves are quite distinct.

The authors found the reaction in all cases up to the fourth week of the disease. It subsided with the acute stage and the gradual improvement of the patient.

TEMPLE FAY, Philadelphia.

THE ARGYLL ROBERTSON PUPIL. S. A. K. WILSON, J. Neurol. & Psychopath. 2:1, 1921.

The Argyll Robertson pupil is not necessarily accompanied by myosis and may be due to lesions other than those of syphilis. The centripetal path for the light reflex is physiologically, and probably also anatomically, distinct from that subserving light perception. The reflex fibers leave the optic tract before the external geniculate body is reached and pass to the superior colliculus by way of the brachium, although the exact path is not known. Partial decussation takes place in the chiasm and again on leaving the colliculus. The convergence and accommodation reaction of the pupil is an associated movement and is activated from the cortex, though the center is not known; and the final infranuclear path is the same as that for the light reflex.

A series of clinical observations is reported to demonstrate the occurrence of an Argyll Robertson pupil in epidemic encephalitis, disseminate sclerosis, tumors in the region of the third ventricle and aqueduct, and syringobulbia. It may also arise from injury to the bulbus oculi and in chronic alcoholism. The most common site of the lesion is in the neighborhood of the aqueduct, but the syndrome may arise from damage to the optic nerve or tract distal to the geniculate bodies. It is suggested that the damage is most often (syphilis and disseminate sclerosis) an ependymitis or subependymitis due to a toxoinfective, lymphatic invasion from the cerebrospinal fluid.

SINGER, Chicago.

ORGANIZATION OF SOCIAL WORK IN A STATE HOSPITAL.
MORTIMER W. RAYNOR, State Hosp. Quart. 7: No. 3 (May) 1922.

Raynor gives the organization of social work in the Manhattan State Hospital in the following stages:

1. At time of admission of patients:
 - (a) Investigations to locate interested friends and relatives
 - (b) Amelioration of home and family conditions
 - (c) Gathering of social history data
2. Follow-up work during hospital residence:
 - (a) Amelioration of home and family conditions
 - (b) Development of plans for leaving hospital
3. Preparole:
 - (a) Investigations and establishing entrée with family.
 - (b) Report to physician
 - (c) Adjusting of home and family conditions for the patient's return home
 - (d) Securing of home or boarding house and a job

4. After-care:

- (a) Supervision through clinics
- (b) Supervision through visits to home and plan of employment
- (c) Advice and assistance in maintaining adjustments and promoting a salutary atmosphere in the home

5. Mental hygiene in community:

- (a) Contact with other agencies doing social work, the schools, and correctional institutions and agencies, etc.

The need of close cooperation and frequent conference between the physician and social worker is clearly stated in this article.

EBAUGH, Philadelphia.

BILIOUS ATTACKS AND EPILEPSY. C. W. VINING, *Lancet* **1**:122 (Jan. 21) 1922.

This author calls attention to the association of bilious attacks and epileptic seizures. In a series of 194 cases in which accurate information was obtainable, seventy-eight patients gave a history of bilious attacks, and ten a history of periodic headaches. In forty-eight cases, these phenomena continued in association with the epilepsy; in twenty cases, they preceded the epilepsy and ceased with the onset of the seizures. Vining, in an attempt to combat the critics of his theory, elicited information regarding bilious attacks from 100 persons attending the public dispensary for conditions other than epilepsy. In this group, twenty-one suffered with bilious attacks, but three of them gave a definite history of epileptic seizures, and eight such a history in near relatives. He also notes a close relationship between nocturnal enuresis and epileptic seizures. The author concludes that some cases, especially in children and adolescents with a history of bilious attacks, associated with headaches or nocturnal enuresis, should be considered as potentially epileptic, and suitable steps should be taken when possible to prevent the development of the convulsive habit.

POTTER, Mercer, Pa.

THE CRAWLING OF YOUNG LOGGERHEAD TURTLES TOWARD THE SEA. G. H. PARKER, *J. Exper. Zool.* **36**:323, 1922.

By placing newly hatched loggerhead turtles in a great variety of environmental conditions and watching their behavior, the following conclusions were reached:

Newly hatched loggerhead turtles find their way from their nests to the sea in consequence of at least three factors: first, their positive geotropism as shown in their tendencies to move down slopes; second, their response to their retinal images in that they move toward regions in which the horizon is open and clear, and away from those in which it is interrupted, and third, their probable response to color in that they move toward blue areas rather than toward those of other colors (Hooker). These animals are not appropriately described as either negatively or positively phototropic, but are to be regarded as exhibiting a more complex condition, in that they respond to the details of their retinal images rather than to these images as a whole.

WYMAN, Boston.

THE CEREBROSPINAL FLUID; ITS SOURCE, DISTRIBUTION AND CIRCULATION. R. M. STEWART, *J. Neurol. & Psychopath.* **3**:144, 1922.

This is a critical review of the literature, and it reaches the conclusions that the only features which appear to be definitely established are: 1. The

fluid is derived chiefly from the choroid plexus and in a subsidiary manner from the perivascular spaces and possibly the membranous surfaces of the brain. 2. Absorption in the brain occurs chiefly through the arachnoid villi and to a lesser extent through the lymph sheaths of the cranial nerves. In the spinal cord elimination takes place chiefly through the lymphatic system. 3. Fluid circulates in the brain ventricles and subarachnoid spaces, but to a limited degree in the spinal spaces.

It cannot be definitely stated whether the fluid is a secretion or a dialysate, the rate of production and absorption, or whether the fluid serves as a lymph or circulates within the brain substance.

SINGER, Chicago.

WAR PSYCHOSES, THE INFECTIVE EXHAUSTIVE GROUP. D. K. HENDERSON, Glasgow M. J. **96**:321 (Dec.) 1921.

This author reports the study of a series of 115 cases in patients invalidated from Mesopotamia, Salonica, India and Egypt. Malaria is given as the cause in forty-four cases, dysentery in 12, enteric fever in 5, heat exhaustion in 13, influenza in 7, septic wounds in 5, and miscellaneous and mixed causes in the remaining 29 cases. Of these patients, 13 were dull, apathetic and depressed; 19 showed hallucinations of a depressive character; 26 were irritable, suspicious, delusional and hallucinatory; 42 were delirious; 7 wandering and amnesic; 3 stuporous; 2 were maniacal, and 3 showed a rather characteristic Korsakoff's syndrome. Henderson concludes that exhaustion as a primary factor in the causation of mental illness has been greatly overrated, although it is of importance as a contributory factor. The symptom picture in infective-exhaustive psychoses varies, and depends chiefly on the innate characteristics of the person.

POTTER, Mercer, Pa.

THREE CASES OF MANGANESE POISONING. J. R. CHARLES, J. Neurol. & Psychopath. **3**:262, 1922.

Of the three cases, two were at first diagnosed as cases of functional disorder, the third as syphilitic myelitis. The diagnosis of manganese poisoning was finally made because all three patients had worked in the same factory in an atmosphere of manganese dust. All presented more or less aphonia, mask-like facies, weakness in the arms and legs with marked spasticity in the legs. Sensation and coordination were unimpaired. The tendon reflexes were brisk, but there was no clonus, and the plantar reflexes were of flexor type. There was no dementia. The shortest duration since removal from the manganese atmosphere was two years, but there was no marked improvement. The damage was therefore considered to be permanent and in all probability located in the region of the basal ganglia.

SINGER, Chicago.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Ninety-Eighth Regular Meeting, Oct. 3, 1922

FOSTER KENNEDY, M.D., *Presiding*

DOUBLE PAPILLEDEMA CAUSED BY BLOCKING OF CORD AT FOURTH CERVICAL VERTEBRA GREATLY RELIEVED BY OPERATION. DR. T. K. DAVIS.

A man, aged 24, with a negative family and personal history, for two years before admission was ill with headache, dizziness and vomiting; he did not have diplopia or fever. He improved and returned to work (clerical), and was well for a year. The headaches then returned, and the legs and arms felt weak. On admission to Bellevue Hospital, in February, 1922, examination revealed slight papilledema diminution of the deep reflexes in the arms, normal reflexes in the legs, a Babinski sign on the left side and normal abdominal reflexes; there were no sensory changes in the upper extremities. A roentgenogram of the spinal column showed no bone involvement or spondylitis, and the roentgenogram of the skull was negative. The spinal fluid was negative for cells and for colloidal gold. Both the spinal fluid and blood Wassermann tests were negative. There was no cervical rib. The youth showed some congenital abnormalities: hypospadias, one undescended testicle, and a doubling up and over of the fifth toe on each foot.

The headaches were greatly improved by lumbar puncture. The patient was kept under observation. Two months later he returned. He had weakness of the arms and hands, marked atrophy of the arms, hands and shoulder girdles and increased headaches and papilledema. The triceps reflexes were now absent; the biceps reflexes were present though weak. There was hypesthesia in the distribution of the third to eighth cervical, and first thoracic roots inclusive. Over the first dorsal vertebra there was also a disproportionate disturbance of temperature sense. On one occasion he had a general convulsion without loss of consciousness, lasting only two minutes. During this there were clonic spasms of all extremities, as well as lateral rotation of the head and eyes from side to side. In view of this convulsion, the severity of the headaches and the increasing papilledema, a laminectomy was deemed advisable, and the third, fourth and fifth cervical laminae were removed in June, 1922. A tense dura was found, with the cord completely filling the dural cavity and an apparent lack of fluid below an enlargement at the fourth cervical segment. Exploration was made for a dural tumor, but none was found.

The swelling about the fourth cervical was incised in the posterior midline, and a cavity was disclosed, which on probing was found to be about $1\frac{1}{2}$ inches (3.81 cm.) long. One interesting feature is that the operator for some reason, not fully explained, was unable to pass a probe upward through the foramen magnum. The patient made an excellent recovery, and the papilledema disappeared. Postoperative treatment has consisted of massage. The greatest disability remaining is in the left deltoid and left pectoral muscles, but even these muscles respond to both faradism and galvanism, and with normal polarity.

DISCUSSION

DR. FREDERICK TILNEY: Did the papilledema disappear after operation?

DR. T. K. DAVIS: The optic cups are still somewhat obscured, and the edges of the disks are a little irregular in outline. These are the only changes, and they probably indicate a certain degree of postneuritic atrophy.

DR. TILNEY: The case appears interesting because the change, so far as one knows, was so remote from the particular lesion. Certain things in the symptomatology, such as the convulsive seizure, would lead one to believe that the lesion in the cervical cord was not the only change in the central nervous system, so that the papilledema might be explained by something else than the blocking of the subarachnoid spaces. When stigmas are so numerous, there may be other lesions, such as embryonic rests or cyst formation in other parts.

DR. FOSTER KENNEDY: The patient seemed to fulfil all the conditions required by our theoretical conceptions. The cervical gliosis was definite, with motor segment atrophies. The segmental sensory disassociation proceeded rapidly, coinciding with intense headache and severe papilledema; this seemed to demonstrate the fact that the papilledema was due to acquired hydrocephalus, and operation corroborated this explanation. The cord filled the canal; there was no pulsation below the level of the bulge. On incision of the dorsal column, a large cavity was found. This upheld our diagnosis in every respect, because the incision of the syringomyelic cavity and cord decompression produced amelioration of the papilledema. That there might be other embryonic defects of the brain which produced the convulsion and papilledema can neither be proved nor disproved, but the facts as we know them are opposed to that conclusion. The convulsion was synchronous with a period of tremendous intracranial pressure, and relief of the spinal pressure produced relief of the intracranial symptoms. These cases of high cervical lesions producing intracranial effects were first recorded in 1902, but this is the first case I have seen.

GENESIS OF CEREBELLAR FUNCTIONS. DR. FREDERICK TILNEY.

This article appears in full on page 137 of this issue.

DISCUSSION

DR. FOSTER KENNEDY: Invertebrates have no cerebellum as we know it, but the postural coordination of insects is perfect. Dr. Tilney's definition of motion as a "fluid stream of postures" is a sound one, and clinicians would agree with that interpretation. Cerebellar lesions produce a decomposition of rhythm, the stream being broken into its component parts. I should like to ask about the idea of the development of lateral portions in birds. I think in some birds, for example, the fighting ostrich and the hawk, there are seen distinct neokinetic powers in the independent action of one limb. Here are highly coordinated movements with almost no development of the lateral lobes of the cerebellum.

DR. I. STRAUSS: There is no doubt about the facts as set forth by the speaker in this painstaking work. Dr. Tilney's thesis involves the discussion of a philosophical question. Does function change morphology, or is morphology altered by something else? The old Darwinian theory of the survival of the fittest has had to be modified. There are myriads of mutations of species. The Darwinian law determines which of the variables should survive; but is it not more correct to say that there have been variables and that certain of these which have been found of use to the organism have been allowed to remain?

That would explain the development of the lateral lobes. In regard to the birds, many of their movements are independent. They are not all associated or automatic. The scratching of hens and the wing movements of some birds in fighting are independent. In the jump from birds to mammals there is considerable lateral lobe development, and there seems to be a gap in the line of reasoning which must be bridged over. Possibly there may be some other factor or force which has to do with development of this part of the brain.

DR. I. ABRAHAMSON: Decerebrate rigidity must be regarded as posture; removal of the cerebellum does not change it; removal of a portion of the brain-stem, especially in the neighborhood of Deiters' nucleus, produces loss of hypertonicity, a change from extensor to flexor postures. This region of the brain-stem is represented in part by the cerebellum bulbare (Tilney) which controls the trunkal muscles, but not the extremities; yet removal of this part causes marked change in the posture of the extremities. I believe that the function of the cerebellum is to regulate or synergize the movements in the passage from one posture to another, that it exerts little or no influence on posture itself.

DR. F. H. PIKE: Sherrington investigated decerebrate rigidity long before the work of Magnus. Decerebrate rigidity appears after transection of the brain-stem in front of the corpora quadrigemina, but the whole midbrain region must be left intact. The rigidity disappears when the dorsal roots of the spinal nerves are divided or when the dorsal and ventral spinocerebellar tracts are sectioned, with the dorsal roots intact. Transection of the brain-stem at the lower border of the midbrain also abolishes decerebrate rigidity. Ninian Bruce traced fibers originating in cells in the nucleus dorsalis (Clarke) up to the anterior and posterior corpora quadrigemina. Thiele had shown, some years before, that careful ablation of the cerebellum did not abolish decerebrate rigidity.

DR. FREDERICK TILNEY: I think there is a definite degree of neokinesis in the bird, which is associated with the incipient lateral lobe formation. There is, however, considerable work to be done before confirming this view. I think posture is an essential of motion. With reference to the philosophic criticism of Dr. Strauss, I think it would take us a long time to reach a definite conclusion, and the discussion would become purely an academic one. With reference to the priority of function or form, we must interpret facts as a form-function relation. I believe that form precedes function, because in passing from one form to another the alteration of an essential part alters the relations of all parts of the body. I cannot answer the question about invertebrates, as I have not made a study of this side of the work. I think we are beginning too high up in our neurologic studies, and shall have to look for the answer to some of our problems in the invertebrates. I am told by those who have studied the subject that there are parts in the arthropods and arachnoids which show cell groupings in communication with the peripheral organs and the semicircular canals, the proprioceptive system and corresponding organs. As far as decerebrate rigidity is concerned, I do not know what it is; no one does. It has not been analyzed.

DEMONSTRATION OF ANIMALS IN WHICH THE RESULTS OF
RECENT LESIONS OF THE CEREBELLAR CORTEX OF ONE
SIDE WERE CONTRASTED WITH THOSE RESULTING FROM
EXTIRPATION OF THE OTIC LABYRINTH OF THE SAME SIDE.

DR. F. H. PIKE (by invitation).

In the cat, the postural disturbance was marked. The head was turned about the long axis of the body, from 45 to 90 degrees to the side of the lesion. If

the animal lay on the right or sound side, the tip of the nose was turned up to an angle of nearly 90 degrees with the table. When the animal attempted to walk, it rolled over repeatedly to the side of the lesion. There was no actual disturbance of the movements of the limbs, but the animal was unable to control its posture or position. It has been shown in other experiments that the head straightens up after division of the dorsal roots of the cervical nerves on the opposite side to that of the vestibular lesions. There are also rhythmical movements of the eyes, with a slow deviation to the side of the vestibular lesion, and a quick movement in the opposite direction.

It was formerly stated in the textbooks that the effects of cerebellar lesions were essentially similar to the effects of vestibular lesions. This was something that everyone knew, and the question was not open to argument. But in the animals (cats) exhibited, those with cerebellar lesions did not show any true ocular nystagmus. Slight oscillations or tremors of the eyes could be seen when the animal looked intently at an object near it. There was no torsion of the head, and the animal did not roll over when it attempted to walk. It turned to the side opposite to the lesion in walking, or went straight ahead. It could turn to the side of the lesion in walking. The fore foot on the side of the lesion was raised higher in stepping than the other fore foot. The symptoms observable were concerned with movement rather than with posture. Our observations on ocular movements in cerebellar lesions are in general agreement with those of Eckhard. He removed the cerebellum of a frog with extreme care, and, in those experiments in which he was positive that he did not injure the medulla oblongata, he did not get ocular nystagmus.

The posture of the pigeon with a severe cerebellar lesion is more affected than is that of the cat with a cortical lesion. There was some rigidity of the legs and toes in the bird exhibited, but this was not indicative of a cerebral lesion, and could not be called decerebrate rigidity. True decerebrate rigidity has not been described in the pigeon. It will be remembered, also, that in Hughlings Jackson's original figure of a child with a cerebellar lesion there was opisthotonos and extension of the limbs.

NEW YORK NEUROLOGICAL SOCIETY AND NEUROLOGICAL SECTION OF THE ACADEMY OF MEDICINE

Joint Meeting, Nov. 14, 1922

FOSTER KENNEDY, M.D., and S. PHILIP GOODHART, M.D., *President*

A COMPARATIVE STUDY OF THE ICEBOX AND WATER BATH
FIXATION METHODS IN THE WASSERMANN REACTION AS
APPLIED TO NERVOUS DISEASES. DR. LEWIS STEVENSON (by
invitation).

The impression has gained ground among laboratory workers that the icebox method has given positive results in syphilis which have not been found with the older methods. Studies of 459 cases of syphilis in all stages made by Wile and Hasley in 1919, showed that the icebox method was more sensitive. Other workers confirmed this view. In the author's work on the blood Wassermann reaction in tabes, which by the older method is usually negative, the icebox method is found more delicate and reliable. It is especially applicable to syphilis of the nervous system, in which the percentage of positive

reactions has been found to be increased about 40 per cent. Perhaps in other forms of syphilis this would not be so marked. It would seem that the icebox method searches out the syphilitic process not discoverable by the water bath fixation method, and, in our experience, positive results were never obtained in nonsyphilitic nervous diseases.

DISCUSSION

DR. DAVID J. KALISKI (by invitation): I have used the icebox method for many years. The facts mentioned by Dr. Stevenson as to the superiority of this method have been brought out by many writers. Incubation in iced water may be even more effective than the dry method. The method is a safe specific one, if not carried on too long. Like other procedures, it has its dangers. If carried on for about eighteen hours or more, there are a definite number of nonspecific reactions. With certain limitations, up to four hours for instance, the method should be used as a routine procedure in all Wassermann test laboratories. There is a possible explanation for the large number of negative results reported by Dr. Stevenson, in cases of tabes, although my experience does not confirm this point. When we performed only water bath incubations, our percentage of negatives was not so low in a large series of cases. One has to take into consideration the amount of natural hemolysin present in different serums. Unless that is considered, the weak reactions would not be detected. In the later stages of tabes, also, a large percentage of negative reactions is obtained with any method. One other statement should be questioned, that is that a negative Wassermann test is looked on as a sign of cure. We all know that a clinician with experience in nervous diseases does not rely on a negative Wassermann reaction in the blood alone as an indication of a serologic or clinical cure.

DR. LEWIS STEVENSON: The technic used in our cases included fixation for four hours, at zero. Negative results were taken as such. Positive results were checked again by the water bath method, and both results reported. The method might be of use in detecting atypical forms of syphilis of the nervous system.

ARTIFICIAL NERVE BRANCHES FOR INNERVATION OF PARALYZED MUSCLES. DR. BYRON STOOKEY.

By growing artificial nerve branches, I have attempted to solve the problem of innervation of paralyzed muscle when its nerve supply is destroyed so that neither direct nerve implantation nor nerve suture are available. Experimentally, I found it possible to make a nerve grow an artificial nerve branch at any desired level by using a free nerve transplant and direct implantation of the latter into the muscle. Branches were grown from nerves at levels where normally branches are not found—such as from the ulnar and median nerves in the arm and to muscles normally not innervated by the nerve selected. After depriving the biceps of its nerve supply, an artificial nerve branch was grown from the ulnar to the biceps. At various intervals, from 120 to 239 days after the formation of an artificial nerve branch, electric stimulation gave a rapid, full contraction of the biceps. The biceps muscle in each was normal in size and color, showing no fibrillations or the yellowish color characteristic of degenerate muscle. Histologic studies revealed normal muscle fasciculi of normal size and with distinct striations. In both longitudinal and cross sections numerous nerve bundles and nerve fibers were found, thus showing that regeneration and innervation had taken place. Tracings and microscopic sections were

shown. The formation of artificial nerve branches had not caused any apparent paralysis in the peripheral ulnar musculature. However, I believe that in the formation of artificial nerve branches some paralysis may result but that this may be minimized by delicate technic and by selecting certain points on the nerve trunk at which to form the branches. I suggest further that the method may be of value in individual muscle paralysis of anterior poliomyelitis, such as the deltoid, the biceps, etc.

DISCUSSION

DR. I. ABRAHAMSON: Was the work controlled by electrical examinations and what information was obtained from such tests?

DR. OLIVER STRONG (by invitation): I have examined some of Dr. Stookey's sections and have seen the bundles of fibers of which he speaks. It is difficult to demonstrate individual nerve fibers in tissues, although nerve bundles can usually be distinguished more easily. In overcoming this difficulty, modern neurosilver stains are of great advantage. Of course, one might use a myelin sheath stain, but where the regenerating nerve fibers have not reached their full state of development they may not have acquired the sheath, and, in any case, the sheath is absent near the termination of the fiber, so that the choice of the silver stain for axis cylinder staining was wise. In this way, one can see individual fibers here and there in the muscles. Those who are not familiar with the histologic side of the work are likely to suppose that we can demonstrate the nerve end plates in this manner, but this is not necessarily the case, as this stain does not always demonstrate the motor plates even when the fibers are stained up to the termination of the latter.

The criticism might be made that these were sensory nerves and not motor fibers. Aside from the improbability that only one type would be regenerated, the answer to this is that the muscle fibers were in an absolutely normal condition, which, of course, would not be the case unless the muscles were receiving the proper innervation.

It is hard to appreciate, even when hearing this presentation, the enormous amount of work that has been given to the histologic side in regard to the regeneration of nerve fibers. This question of nerve regeneration is important, and there are two principal theories as to the way in which it takes place. One of these theories is that of centrogenesis, according to which the regenerating fibers are outgrowths of the central stumps; and the other is that of autogenesis, according to which the nerve regenerates in situ. The neurolemma cells of the distal stump proliferate and form what are termed band fibers. The autogenists believe that the new nerve fibers are formed out of the substance of the band fibers.

By means of the silver staining methods the axis cylinders, especially the regenerating ones, can be clearly demonstrated. The clearness of the results obtained by this method is shown in the picture of the cross sections of regenerating nerve fibers, which by no means exaggerates the amount of detail which can be made out and the clearness of the preparation. The application of this histologic method shows that the new fibers are outgrowths from the central stump. The band fibers which are formed by the old neurolemma sheaths are important in establishing the old connection with the central nerve fibers, but the regenerating fibers are not formed out of them but grow along or in them from the central stumps to their appropriate destinations. The sprouting nerve fibers, for example, traverse blood clots or enter muscle tis-

sue in which there are no band fibers. The fibers entering the scar tend to branch and thus find their way across the scar to the band fibers on the other side. By following the band fibers they are found finally to reach the vicinity of their former terminations in the muscle fibers. When they reach the vicinity of the old motor end plate possibly some chemotactic influence of the remnants of the old motor end plate attracts the motor fibers to their proper terminations in them.

Some interesting work has been done by Jello and others on the innervation of the motor end plates in the embryo. From this it would appear that they reach the embryonic muscle fibers and come in contact with them before the sarcolemma is formed. This explains the trypolemmal location of the end arborizations and possibly the profound influence of the nerve fiber on the muscle fiber. There is evidently some relation between the nerve fiber and the nucleus of the embryonic muscle fiber. The nuclei of the embryonic muscle cells are central, but later in the course of development they become superficially placed. The growing nerve fibers in the vicinity of a muscle nucleus show thickening. They then arborize and as they arborize the muscle nuclei in their proximity proliferate, thereby forming multinucleated "sole plates."

In regard to the practical application of these latter observations, possibly accelerating chemical substances might be used to aid regeneration. Chemotactic influences probably form an important part of the rôle played by the band fibers and also by the pieces of resected nerves used in Dr. Stookey's experiments.

DR. LOUIS CASAMAJOR: This work is a combination of nerve graft and nerve implantation. The muscle in these cases had had no chance to degenerate. Such a formation of branch is possible, but further work is necessary to show whether a similar success can be obtained in a muscle which has already undergone atrophy. In our operations, we usually encounter muscle which has already become atrophic. These particular experiments should not be applied to the human subject, because we should not care to sacrifice part of the ulnar to get a good biceps. A man could get along with a poor biceps, but he could not manage without a good hand. In the paralyses of poliomyelitis, this method would have a definite application. Muscle transplants have been fairly successful, but the results have not been fortunate. We might obtain neuronization of the muscles, and a great deal might be added to the happiness of the individual. Part of the posterior tibial could be sacrificed, which would be quite justifiable if this could be done after the muscle had degenerated. There might be some return of function, which is the greatest hope that can be held out in these cases.

DR. ALFRED S. TAYLOR: I remember the work done on nerve transplant by Dr. Globus six or eight years ago. He took a segment from a good nerve and implanted it into muscle; the work was not so interesting as this, nevertheless it gave good results. The muscle gave good contractions when the nerve was stimulated. Good end plates were seen clearly in slides from the muscle. I think Dr. Stookey would find them in his muscle if he searched long enough. With regard to dividing a small segment of nerve and not finding paralysis, an Englishman, working with the hypoglossal anastomosis, found that one third of the hypoglossal nerve could be divided without causing paralysis. Other investigators have shown that the fibers of a nerve branch are collected 2 or 3 cm. above its exit from the main trunk, and above that distance the fibers pass freely from one bundle to another. If the nerve trunk is divided

at any point well above a branch, it is conceivable that the nerve damage involves axis cylinders going to any one branch, and the damage is so diffused that peripheral failure occurs.

In regard to poliomyelitis cases, I am rather pessimistic. I performed a number of nerve transplants in such cases twenty years ago. In one or two, there was partial regeneration; in eight or ten flat failure. In the case of a child very well cared for, who had received massage and other treatment for four years after paralysis of the peronei, the neurologist recommended nerve transplantation of the peroneal nerve into the internal popliteal. After six months, there was distinct evidence of regeneration by electric stimulation, but the functional result was never satisfactory. The child could evert the foot slightly, but enough function could never be restored to make it useful. Drop foot persisted, although there was good voluntary movement in the muscles. I would not advise operation in these cases. Poliomyelitis is more or less of a group paralysis and there may be sufficient damage to neighboring nerves to prevent one obtaining a good virile source to tap for a nerve supply when anastomosis is attempted. This is one reason for failure in these cases.

DR. CHARLES A. ELSBERG: Experiments of this kind in the laboratory will be of assistance to the surgeon. One point I would like to emphasize is that such experiments require an extreme delicacy of technic, which few workers have attained. These fine nerves must be handled gently to avoid damage. Many experiments in the past have failed, or given variable results, because of lack of delicacy in the technic. Another point is that from the gross appearance of the muscle and the muscle contractions there was satisfactory evidence of regeneration even without histologic examination. The muscle deprived of its nerve supply presents a characteristic appearance in the gross. When the muscle is again supplied, its appearance is entirely changed, and it looks just like normal muscle and cannot be distinguished from it. With electrical tests they give the same contraction. The question has been raised as to what would have happened if these muscles had been atrophied. Clinically, we know that old facial paralyses return to normal. Muscles partly atrophied have a hunger for nerve fibers. If Dr. Stookey had been dealing with muscles deprived of nerve supply, the results would have been more striking. There is a good outlook for the application of this work in some muscular paralyses, whether due to poliomyelitis or to other causes.

DR. M. A. KRAUS: No one can question the great importance of these studies from either a theoretical or a practical point of view.

There are two points in this connection which I should like to emphasize. Dr. Stookey is aware of the difficulty of determining the fascicular content of a nerve at a given level. Furthermore, there are many different kinds of fibers at a given point—motor, vasomotor and various sensory types. A distance of 2 cm. might make a good deal of difference in the fascicular content.

The second point is that the work of McKinley of the University of Minnesota, mentioned by Dr. Taylor, must be considered carefully before it is accepted. In my opinion, the conclusions are not all proved by the experiments. Indeed, many of his conclusions seem to contradict his findings. The statement that the fascicular picture on cross-section at various levels of a nerve varies a great deal, is undoubtedly true, but the fibers which make this continual change, in the vast majority of cases, have different functions, such as motor, vasomotor and sensory—not the same function; in other words, there is a continual gathering into a few fasciculi of fibers of motor, sensory and vasomotor

function preparatory to the leaving of these fasciculi as a branch from the main nerve trunk. It follows naturally that there is continual splitting of fasciculi having various functions.

Since McKinley does not note the entire physiologic significance of each fasciculus, his conclusions in regard to motor fasciculi do not bear careful scrutiny. Indeed, some of the fasciculi he describes are those to joints. Second, the experiments in cutting the sciatic and the single experiment using electrical stimulation were performed from 3 to 4 cm. below the sacral plexus between the ischial tuberosity and the great trochanter of the femur, a point so near the plexus that changes in the fascicular topography of motor fibers immediately below would naturally be expected. The peripheral combinations from various segments going to muscles must occur distally to the plexus. I do not believe that the work of Marie, Meige, Gosset, Déjerine and M. and Mme. Mouzon, not to mention that of Dr. Ingham and myself, can be thrown out so lightly.

This all serves to emphasize some of the difficulties of using nerve branches in practical surgery. It does not in the least detract from the great importance of Dr. Stookey's researches in paving the way which will overcome these difficulties.

DR. STOOKEY: Replying to Dr. Abrahamson, we did not use any electric stimulation during the progress of the experiments. There is no method, other than that of Lapique's chronaxie, to determine the downgrowth of the neuraxes; and this method is not feasible in animals. We stimulated the biceps muscle in the operative field during the progress of the studies in two animals. I have no other data concerning the rate of the downgrowth.

Dr. Casamajor properly raised the question as to the wisdom of going into the ulnar nerve to supply the biceps. We used the ulnar nerve and the biceps muscle experimentally, because this seemed to offer the best technical opportunities in the dog. In man, the choice of nerves and the decision to make an artificial nerve branch depends entirely on the paralysis and the results that may be gained. Obviously, it would be unwise to induce a paralysis of the intrinsic muscles of the hand in order to obtain innervation of the biceps muscle. In reply to the question about innervation of old paralyzed muscles, I would refer to the fact that paralyzed muscle regularly regenerates if neuraxes are supplied, providing too long a time has not elapsed and degeneration of the muscle is not too far advanced.

I am aware of the work to which Dr. Taylor called attention. A flap is raised from the nerve and implanted in the muscle. I would like to emphasize the fact that in making such a flap from the main nerve trunk damage to the nerve is many times greater, and the possibility of successful repair is diminished; but with a fine nerve segment, an artificial nerve branch may be made any length desired with infinitely less trauma to the nerve trunk.

Dr. Kraus mentioned Stoffel's work on funicular anatomy. I do not believe there is a definite funicular anatomy any great distance from the point at which the nerve branches arise. The funiculus to the pronator teres is almost a definite nerve within the sheath of the median. Other examples are to be found in the sciatic, but with these exceptions I do not believe a distinct funicular anatomy exists from the origin of the nerve to its peripheral distributions. Langley and Hashimoto have shown that throughout the course of the sciatic nerve, nerve plexuses which rearrange the nerve fibers are numerous. Borchardt and Wjasmenski (*Beitr. z. klin. Chir.*, 1917 and 1919)

have shown by elaborate studies and dissections on especially prepared nerve trunks, using macerated specimens, that there are no definite funicular paths from the origin of the nerve to the periphery, but that there is a constant interlacing and innumerable nerve plexuses. I believe the only funicular anatomy found is immediately above the origin of nerve branches, where there is a shunting of definite neuraxes on the formation of the nerve branch about to be given off. In certain nerves and certain positions, such as the musculo-spiral in the middle third of the arm, the small funiculi are grouped together into one large bundle. In selecting the points for formation of artificial nerve branches an intimate knowledge of the finer anatomy of the nerves concerned is presumed.

Dr. Taylor mentioned what is apparently anatomic regeneration without functional regeneration. Complete functional regeneration depends on the afferent proprioceptive impulses as well as efferent impulses. I remember a tailor, who, during the war, had a median nerve suture performed. There appeared to be complete regeneration, but the man did not regain function so as to be able to return to work. He could hold his scissors but could not use them accurately, probably due to a loss of the muscle tendon sense; and he had what could be termed a peripheral apraxia.

A CRITICAL REVIEW OF THE THEORIES OF THE PATHOGENESIS OF EPILEPSY, WITH A NEW INTERPRETATION OF THE AVAILABLE DATA. DR. MICHAEL OSNATO.

The various important theories of the pathogenesis of epilepsy were briefly discussed. An attempt was made to construct synthetically a conception that would harmonize all the available data. The chief feature in the conception offered was based on the work concerning the biochemistry of the blood in epilepsy.

Briefly summarized, this work calls attention particularly to the defective metabolism of the starches in epileptic persons. Organic acids, chiefly acetic, butyric, lactic and tartaric acids, are formed during the second stage of the breakdown of starches by the amylolytic ferments. In normal persons, these acids are changed by the action of the liver and the small intestine into salts, and they are then oxidized into ammonium carbonate or sodium carbonate and urea. In epileptic patients, however, these acids are found unoxidized in the urine and the blood.

It has been proved that in an acid medium, the nucleohiston content of cellular nuclei is broken down into nucleinic acid and proteoses (albumoses). These substances cause the convulsions of the epilepsies.

The acids mentioned, especially lactic acid, disturb the vasomotor tone of cerebral blood vessels. In the generalized convulsions, the unconsciousness is caused by the sudden vascular shutdown, when these toxic substances reach the brain.

In the smaller jacksonian type of seizures, the production of these substances (proteoses) may occur locally in the brain tissue or may be imprisoned locally by sharply circumscribed vasoconstriction.

DISCUSSION

DR. LOUIS S. ARONSON: It is of interest to note that borax as a remedy for epileptic convulsions was put forth by Gowers many years ago. During the recent war, the French had great difficulty in obtaining phenobarbital (luminal,

or gardinal as they call it), and in order to avoid bromid therapy, attempts were made to return to some form of borax for the control of the epileptic seizures. Marie and Bouttier, after trying potassium and also sodium tetraborate, obtained good results with potassium borotartrate. This is not a mixture; it is a chemical which apparently has some direct effect on intestinal chemistry and the fundamental reactions that have been alleged to be the basis of the convulsive attack. The authors of this form of treatment assert that following its use for several months there is in a given number of cases a complete cessation of convulsions and in the great majority of cases a marked diminution in the number and severity of the attacks. Most patients have 50 per cent. fewer attacks, and many of the attacks are converted from major into attacks of petit mal. Marie and Bouttier do not claim any special cure with this mode of treatment, and, in fact, administer a small dose of phenobarbital to many of their patients once daily, when tartroborate of potassium fails them. They claim, however, that this preparation can replace the bromids and accomplish as good results as the bromids as an adjuvant to phenobarbital, without producing the undesirable acne, indigestion, impoverished memory and weakened sexual function that one so frequently finds in the chronic bromid user. I have seen many of their patients that were free from attacks for nearly two years on borotartrate medication alone, and many that were benefited to a considerable degree and happy that they were not being given bromids. My own experience is limited to the use of the drug for the last four months, since I have succeeded in obtaining some from Paris, and all that I can say for the present is that it seems to control some of the cases. It is an extremely hygroscopic crystalline flake that cannot be prescribed other than in small vials of one-half to two drams (1.37 to 7.5 c.c.), two to three times daily, for the patient to dissolve and drink in fresh solution.

DR. FOSTER KENNEDY: The high degree of carbohydrate fermentation found in the intestine is of much interest. The relation to intoxication is more probable than to nervous instability. The fact of deprivation of the blood supply seems to be the chief factor. I tried on myself the experiment of compressing both carotids, and before I could count two lost consciousness. The great underlying factor is that of vascular instability. The causes are faulty chemical processes. I believe there is only a difference of degree between the ordinary syncopal attack and that of the epileptic convulsion. The phenomena have the same underlying basis, namely, vasomotor disturbance.

DR. S. P. GOODHART: Did Dr. Osnato consider the aura as part of the epileptic seizure?

DR. OSNATO: In reply to Dr. Kennedy, fifteen years ago, Pike tied the internal carotids and the vertebral close to the subclavian, in cats. Asphyxia and a tonic spasm immediately ensued, having all the characteristics of decerebrate rigidity. Artificial respiration continued during the ten minutes that the occlusion was allowed to continue. Immediately, and for a period of four or five days, the cat would have a series of convulsions, which could be brought on even by stroking its fur, jarring the table or making loud noises. This experiment is explained in view of the biochemical studies of Cuneo, by the fact that when cells are shut off from their blood supply, immediate catabolic changes occur. The nucleohiston breaks down into nucleinic acid and proteoses in the presence of the acid medium supplied in this particular case by the asphyxiated blood.

I am quite certain that epilepsy is not a brain disease, that is, that the disease could not be explained by investigation inside the skull alone. I have

carefully studied fifty-seven cases of epilepsy and found that in only three were there any objective neurologic signs. Two of these cases showed sensory motor types of cortical disturbances. They were traumatic cases with craniocerebral injury. The other case was one of so-called idiopathic type and showed a Babinski sign on one side and a sixth nerve involvement on the other.

Although private cases were few in number, I chose them because they were much better material than larger groups of cases from a clinic. Even in good neurologic clinics, patients are examined only once, usually by men whose training neurologically has not been completed. There are also many other sources of error in the examination of clinic patients. However, I have no doubt that if it were possible to examine thousands of cases of idiopathic epilepsy, the numbers showing actual objective neurologic signs would be few. I believe, therefore, that epilepsy is not primarily a disease of the nervous system, but makes itself felt through it in the convulsion and some other episodes.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY*

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F. H. PACKARD, M.D., President, in the Chair

TWO CASES OF DYSTONIA LENTICULARIS. DR. H. C. SOLOMON.

There is no doubt as to the diagnosis of dystonia lenticularis in the first case. The important point is that the first symptom was a peculiar contracture of the muscles of the left foot. This caused the patient to walk with an unusual limp; but he was able to run in a normal fashion. Therefore the diagnosis of hysteria was made. This condition continued for about three years and then spontaneously disappeared for a period of six months, when it reappeared. From then on progress was rapid in all parts of the body except the head. The patient developed the characteristic symptomatology of dystonia lenticularis, including torsion of the body, hypotonic and hypertonic condition of the muscles, and marked disorder of equilibration, which, however, never led to falling. After a time, a characteristic dromedary gait developed, and later the patient was unable to walk. The second point of interest was a partial remission, so that at the time of presentation the patient, after having been confined to a chair for many months, was again able to walk and go about fairly successfully.

The second patient was a young lad of 17, who about two years previously rather suddenly developed spasmodic torticollis. Six months before the development of this condition, he had been slightly injured in an automobile accident. A few days before the torticollis appeared, he had had two convulsions, which were the only convulsive phenomena that had occurred. Various therapeutic devices had been attempted for a year, including electricity, massage, gymnastics, a plaster cast, a Thomas collar, reeducation, suggestion, psychologic analysis, suggestion under ether, etc. Slight temporary improvement had followed many of these procedures, but on the whole the condition had become progressively worse.

The patient presents a spasm or contracture of the trapezius and sterno-cleidomastoid muscles of the left side, with occasional relaxation. A consid-

* Communications from members of the staff of the Psychopathic Hospital, Boston.

erable degree of scoliosis is present. Occasionally the patient is able voluntarily to straighten his neck. At other times, this occurs spontaneously. There has been a considerable degree of myodemia, which is not now present. The biceps and triceps reflexes are very weak. The other reflexes are active. All the muscles are irritable to percussion; otherwise the neurologic and physical examinations are negative.

It is suggested that this is possibly a case of dystonia lenticularis with the onset of symptoms localized largely in the muscles of the neck, but slowly progressing, the patient having already developed a certain degree of torsion. It is realized that it is not possible to make a definite diagnosis of dystonia lenticularis at this time. There is no method of making this diagnosis, except that of the symptomatology. As shown in the first case, it is possible to have symptoms localized in one part of the body continue for a considerable period before a more generalized spreading takes place; a remission of a greater or lesser degree may occur; considering the onset, the disease may well be considered as hysteria.

The most frequent locus of symptoms at the onset is the leg. Dr. E. W. Taylor has presented a case beginning in the arm and a probable case beginning in the neck. It is suggested that this is a case of dystonia lenticularis with the early symptoms affecting the trapezius and sternocleidomastoid muscles.

DISCUSSION

DR. E. W. TAYLOR: In the first case reported, which I have had an opportunity to observe, there has been improvement in the last two weeks. I might add that the patient is extremely euphoric, and is convinced that he will be entirely well and wishes forthwith to begin work at an art school. In this case, there cannot be the slightest doubt as to the diagnosis. The patient shows, in peculiar degree, the characteristic feature of dystonia, which is a combination of hypertonia and hypotonia, as its name implies. If one notices carefully the way he holds himself, one sees that some of the muscles are in a spastic state, while others are in a state of hypotonia. It is characteristic that he has never fallen in the ten years during which he has been afflicted. There is no Romberg sign; he can stand with his eyes closed without reeling. This is an addition to the literature on this rare disease, which a few years ago numbered not more than thirty or forty cases.

Regarding the second case, I should think the ultimate diagnosis would depend on whether or not the condition spreads from the neck muscles. It appeared to me that probably the patient had a torticollis. The sternocleidomastoid and trapezius muscles were markedly involved. With this symptom alone one would hardly be justified in calling the condition dystonia; but the process has already begun to extend to the muscles of the body, as shown in the distortion of the trunk. I should not regard the condition as hysterical; it is probably dystonia, but up to this time it can hardly be distinguished from a marked case of torticollis. Is it not probable that the movements of torticollis may be due to a lenticular lesion, or at least to a disturbance of function in that part of the brain?

TWO CASES OF PELLAGRA. DR. S. K. SMITH.

I am presenting two cases of pellagra to emphasize the apparent increasing frequency of the condition in the New England States. There are about six cases in Boston at the present time.

The first case is that of a Scotch woman, aged 43, who gave a history of chronic alcoholism. The patient had eaten little meat for a year and probably

had used no milk. There was a history of mental change of three months' duration, together with glossitis, diarrhea, a bilateral dermatitis of the hands and a loss of weight of two months' duration. Examination revealed (1) marked emaciation; (2) negative neurologic symptoms, and (3) typical delirium, with complete disorientation, confabulation, memory changes and hallucinations.

The second patient was a man of 38, who also had a history of chronic alcoholism. His dietary history suggested a poorly balanced diet. For about three months there had been: (1) alternating gains and losses of weight; (2) alternating constipation and diarrhea; and (3) dermatitis, starting as a macular eruption. Examination revealed: (1) marked emaciation; (2) characteristic dermatitis of the hands, feet, elbows, forehead and neck; (3) neurologic findings which showed sluggish pupillary reflexes, absent abdominal and absent right Achilles reflexes; (4) simple mental deterioration, with marked memory change, disorientation, confabulation and hallucinations.

A CASE OF DEPRESSION WITH FEELINGS OF UNREALITY. DR.
C. H. MORRIS.

A married woman, aged 26, gave the following account of the day during which she had lost the data of personal identification: "I left my house in the morning to go to church. Everything at home seemed natural. In church everything seemed different. I did not understand what the priest was saying. When I came out of church I wandered around and got on a car. I think I changed cars several times. I did not know where to go. I could not remember where I wanted to go. I could not remember who I was. I sat all day in a field near Randolph. I knew I couldn't stay there, but I did not know who I was or where to go. Then I walked around and then rode on cars some more. I think I was over in Cambridge, and I think it was 8 o'clock when I remembered my telephone number and everything came back. I called up my sister and told her I would come right home. I came out—I intended taking a car, but forgot again. I walked up and down streets and then everything came back and I went right home. I think it was about eleven when I got home."

How are we going to interpret this loss of the data of personal identification? In this episode it appears that the loss was not in the setting of a delirious condition, not in one of disorientation, for the patient was able to take cars, to go to different places, and to do this without attracting attention. The relation of the loss of personal identification to mood is interesting, for though in many cases of depression the patient feels a change in personality, feels that his actions are no longer self-directed, and that the world has lost a familiar quality, this does not lead to an amnesia of name and place. There are other cases besides those of depression in which there is a peculiar alteration in the feeling of personal identity: those cited by Janet and termed *psycholeptic crises*, in which a patient may have a feeling that he is different, that is, "It is not I who eats, speaks, works. I lack something to give me real existence." Here we have no loss of the data of personal identification, but rather a change in the feeling tone which is at the basis of personal identification. In other cases of amnesia, the patient in a new environment has forgotten all that identifies him with a painful past environment.

None of the types mentioned in the foregoing quite fits the present case: neither a severe depression, depression with feeling of unreality, *psycholepsy*, nor hysterical type of reaction. The clinical picture is that of a married woman of 26, who had a bereavement in the early part of January, 1922, in the loss

of her third child, at the age of 2 weeks, and who soon afterward developed a pleurisy with effusion. In February, she received a letter from her brother in Colorado warning her of the danger of pleurisy and telling her that it was usually due to tuberculosis. Her brother died of tuberculosis soon after this. In March, she had pneumonia and claims never to have recovered, for she has since had an empty feeling in her head, poor appetite and palpitation. In March, while recuperating, she began reading a medical book with especial reference to tuberculosis and insanity (her mother had died in an insane asylum two weeks after having pneumonia). After reading about hallucinations, she heard a chain dragged across the floor, and knocking on the windows. Since March she had been depressed, had taken little interest in anything, and had been retarded. She entered the hospital in July, presenting at that time a picture of depression and retardation, and did not develop a feeling of unreality until her first visit home in September. On that occasion she said that the home looked "dark and dreary and different from the way it used to." She described her second visit a week later as follows: "I did not know what it was all about in church, but I knew I was in church. I saw all the people and priest, but I didn't know why they were there. At home everything looked queer and mixed up as though the tables and chairs didn't belong there. I could not think." On her third visit home, the incident of the loss of the data of personal identification occurred, and on that same day her two children were found comatose, and one died later at the City Hospital, where a diagnosis of veronal poisoning and bronchopneumonia was made. The children had been alone with the patient on the preceding day. On her return to the hospital she did not remember having been there before, though she had been away five days only, and said: "Everything looks queer. Nothing looks natural. People look as though they were pieces of wood walking around." At first she did not recognize people, and later said that her husband talked about things she used to know about. In this case we have both a feeling of unreality and the loss of the data of personal identification.

In the etiology we have a serious illness from which the patient claims never to have recovered fully, worry over her brother's prognosis in regard to tuberculosis, and worry over the restriction of her family, which seemed necessary on economic grounds and yet which could not be reconciled with the teachings of the Catholic church. The physical status does not show a fully adequate basis for this reaction, nor is this psychosis of the same type as a depression of endogenous origin; this difference of type suggests a different etiology.

DISCUSSION

DR. F. H. PACKARD: It would seem to me that this patient was one who simply began to become confused and dazed, and in the beginning events began to be vague. It is a little hard to understand how, at this time, she could remember what happened when she could not remember anything. I should say the explanations she makes at the present time are merely attempts to explain a situation.

PRESENTATION OF FIVE CASES OF MENTAL DISORDER WITH LOW BASAL METABOLISM. DR. KARL M. BOWMAN.

CASE 1.—An unmarried woman, aged 44, who was admitted to the hospital in a stuporous condition with a history of a cold three weeks before admission, and weakness, became excited and noisy, and finally developed a stupor. The basal metabolism was —56 per cent. As far as known, this is the lowest basal

metabolism on record. Examination revealed a typical case of myxedema. Attention is called to the sparse hair, scanty eyebrows and pubic hair, and lack of axillary hair; the dry, rough, leathery skin with a yellowish tint; the marked edematous and flabby eyelids, and the large, beefy tongue. The urine showed albumin and casts; the phenolphthalein excretion in two hours was 50 per cent. The blood sugar curve was: fasting, 134 mg.; one hour, 144 mg.; two hours, 160 mg.; three hours, 170 mg.

In contrast to the first case in which the low basal metabolism was thought to be due essentially to hypothyroidism, four other cases are presented in which it seems impossible to demonstrate any such cause.

CASE 2.—An unmarried girl, aged 22, had been having narcoleptic attacks for the last nine years. The basal metabolism in this case was -18 per cent.

CASE 3.—A girl, aged 10, had had typical grand mal and petit mal attacks since she was 10 months of age. The basal metabolism in this case was -24 per cent.

CASE 4.—Conversion hysteria was diagnosed in a married woman, aged 40, who for the last year and a half, following some distressing incidents, had developed fainting attacks and typical hysterical anesthesia and paralysis of the right arm. The basal metabolism in this case was -26 per cent.

CASE 5.—Schizophrenia was diagnosed in a boy aged 15, who had developed a stuporous condition about a year before, in which he was mute, showed some waxy flexibility, had hallucinations at times, and had to be fed by tube. The condition had followed a blow on the head. The basal metabolism in this case was -20 per cent.

The first case is presented because it is typical of myxedema and probably shows the effect of lack of thyroid secretion on basal metabolism. In the other four cases, the cause of the low basal metabolism is unknown. Careful studies have been made in every case for evidences of endocrine disorder, and the effects of various endocrine feedings are being tried without satisfactory results.

Further studies in basal metabolism at the Boston Psychopathic Hospital have demonstrated a marked tendency to low basal metabolisms in various types of mental disorder. No attempt can be made at present to explain this, and the cases are presented merely to call attention to an interesting clinical observation which may lead to further study, and perhaps give us some further insight into the mechanisms of mental disease.

DISCUSSION

DR. PERCIVAL BAILEY: Dr. Bowman says he has no explanation to offer for the low metabolism. I also have no explanation, only an hypothesis. It seems to me unnecessary to look further than the brain for the lesion responsible. There is in the base of the brain—to be exact, in the hypothalamus—a region which exercises control over manifold visceral and chemical activities in the body (metabolism of sugar, proteins, heat regulation, etc.). It seems to me reasonable to seek the explanation of both the mental disturbances and the disturbances of metabolism in these cases in the same pathologic influence on the brain.

THE ACTUAL ENVIRONMENT OF CHILDREN IN A BOSTON DISTRICT. DR. MARIANNA TAYLOR.

A survey has recently been made of a section of Boston by the Mental Hygiene Association from the point of view of the mental health of the children.

Fifty homes of all types were visited—also kindergartens, day nurseries and clinics. The findings were exceedingly significant.

In an unselected series of 190 children, the following conditions were noted:

1. Speech Defects	
Baby talk	10
Stuttering	1
Late in talking.....	4
Mutism	2
2. Motor Habits, Incoordination, and Convulsive Manifestations	
Fidgetiness and choreiform movements.....	13
Twitching of eyes and face.....	7
Shivering	2
3. Incontinence, etc.	
Bed wetting	49
Incontinence by day.....	27
Soiling	9
Difficult micturition	1
4. Conduct Defects	
Holding of breath.....	1
Tantrums	52
Spoiled child (stubborn, difficult, uncontrolled).....	41
Excessive boldness	11
Excessive timidity and shyness.....	13
Excessive finnickiness and precision.....	1
Runaways	3
Truancy	2
Pilfering	1
Biting and scratching others.....	5
Seclusiveness	6
5. Sleep Disturbances	
Restless sleep	12
Disturbing dreams	7
Sleep walking	1
Crying out in sleep.....	6
Sighing in sleep.....	2
Talking in sleep.....	11
6. Mental Habits and Mental Grades	
Mentally retarded or defective.....	13
Children in one grade two years.....	18
Day dreaming and fantasizing.....	3
Preoccupation and absent-mindedness.....	4
Epileptic children	3
Idiocy	4
Mentally deficient, epileptic, or psychotic parents.....	18
7. Miscellaneous	
Capricious appetite	17
Headaches	2
Imitateness (excessive)	1
Masturbation	12
Thumb and finger sucking.....	10
Nailbiting	33
Picking of nose, lips and fingers.....	26
Gritting of teeth.....	3
Strabismus	10
Excessive jealousy	2
Hydrocephalus	2

The actual environmental factors and neurotic traits found can best be appreciated by citing a few illustrative cases:

CASE 1.—In the home of an Italian laborer the six children, ranging in age from 10 years to 10 months, were found to have all been slow in developing, none talked before 2½ years of age, one is retarded at school, three have enuresis, all masturbate. The 5 year old child set herself on fire recently and was seriously burned; the mother became maniacal and died within two weeks in an asylum.

CASE 2.—In an American family consisting of father, mother and three children, the father had recently been a patient in the Psychopathic Hospital for eighteen months. He had a violent, uncontrolled temper, and his wife refused to have him placed in an institution. The oldest child showed many neurotic traits. She was pale and undernourished. She was so morbidly anxious to be on time at school that each night she put on fresh clothes and slept in them in order to be punctual the following morning. The second child had had convulsions several times, and the third child was premature.

CASE 3.—In a family consisting of four girls and one boy, the oldest presented a number of neurotic habits; for example, nail-biting, talking in her sleep and picking her nose. She and one sister were retarded at school and had repeated a grade. Two other children had enuresis, day and night; no effort was being made to correct these conditions.

CASE 4.—The next family consists of father, mother and eight children ranging in age from 16 years to 5 months. The 15 year old boy had enuresis, the 5 year old girl bit her nails, and the 3 year old child sucked her thumb constantly. She attacked her sister frequently with a butcher-knife. The 16 year old daughter recently ran away and married a man much her senior, because her mother beat her, gave her no freedom and made home unbearable.

CASE 5.—The next case is that of an epileptic mother who beat her children and spent the greater part of her time on the streets, leaving her children locked in, in charge of the 5 year old child. One of them was retarded in school, had a speech defect, was a finnickier eater, and was underweight. One slept poorly and had convulsions.

Such findings point to the need for educational and preventive work along the lines of mental hygiene: the establishment of mental hygiene clinics, the instruction of social workers, nurses, and parents, and better recreational facilities and supervision of the juvenile population.

THE CHICAGO NEUROLOGICAL SOCIETY

Nov. 16, 1922

HAIM I. DAVIS, M.D., *Presiding*

AN UNUSUAL SYNDROME: ANOSMIA AND AGENSIA. DR. H. DOUGLAS SINGER.

A medical student 26 years of age, eight weeks ago was thrown from a truck, striking his head in the region of the lambda. The accident happened on a Thursday afternoon, and he remembered nothing before the following Saturday

morning. He had a little headache for a few days, but was able to return to school on October 1, about two weeks after the accident. At that time he had a slight headache, tired easily and noticed that a strong light "bothered his eyes." These symptoms disappeared after a short time. Since the accident he has experienced loss of the senses of smell and taste, at first absolute. Taste is beginning to return slightly, and he can distinguish food which is salty, but there is no sense of smell. He constantly experiences an unpleasant odor, which he compares with that of xylol.

Roentgenograms of the skull show a starred fracture at the point of impact in the apex of the occipital bone just behind the union with the parietal bones and a broad fissure extending forward as far as the coronal suture, completely separating the two parietal bones. On both sides, there is also, for a short distance, some separation of the fronto-parietal sutures. The interesting question is as to the seat of the lesion causing this loss of taste and smell without other symptoms. The combination suggests that it must be cortical and, of course, bilateral. The visual fields are normal; fifth nerve sensibility in nasal and buccal cavities is intact, and there is absolutely no other evidence of damage. There is no history of hemorrhage from the nose or ears.

TWO CASES OF BRAIN TUMOR WITH VENTRICULOGRAPHY. DRs.
PETER BASSOE and CARL B. DAVIS.

This article is published in full on page 178 of this issue.

DISCUSSION

DR. T. D. ALLEN: In the first case the muscle findings were not very satisfactory, and the atrophy suggests that there was some pressure irritation or even pressure obstruction of the nerves. It was interesting to find a segmental defect in one eye of this boy. I did not take muscle measurements in the second case. So far as I recall, there was no evidence of paralysis of any muscle, but one would expect from the section that there should be paralysis of some ocular muscle, just as there seems to be pressure atrophy of the second nerves.

DR. GEORGE L. DAVENPORT: I should like to ask one or two questions: First, was the ventriculography performed under local or general anesthesia?

DR. PETER BASSOE: General.

DR. GEORGE L. DAVENPORT: Were there any brain findings postmortem, to which death might be ascribed?

DR. H. A. OBERHELMAN: None except those mentioned by Dr. Bassoe—increased intracranial pressure.

DR. DAVENPORT: My experience embraces over twenty cases, and in a few of these the injection of air has been repeated. Local anesthesia is used in all, unless the patient is extremely nervous and will not cooperate; then general anesthesia is resorted to. The pictures are taken soon after the injection of air, and it is hard to get clear ventriculograms, owing to the movement of the head caused by the forced respiratory movements when the patient is under, or coming out from the influence of, a general anesthetic.

I have not had a death that can be ascribed to the ventriculography procedure. I might qualify that statement by saying that one patient died suddenly eighteen hours after the operation. Dr. LeCount, at the necropsy, found no

reason for assigning death to the ventriculography procedure. There was a large cerebellopontile angle tumor and we felt that the sudden death of a patient with a tumor of this size and in this particular location was not at all unusual, even when operative procedure is not used.

Possible causes of death from ventriculography are: hemorrhage into the ventricles, air embolism, sudden reduction or increase of intracranial pressure, rupture of a vessel, or the sudden displacement of some vital area.

In regard to Dr. Bassoe's cases, I believe the imperfect results can be ascribed to faulty technic, the ventricles not being satisfactorily emptied and refilled with air. All my work has been done through Kocher's point, entering the ventricle at the junction of the anterior horn with the body of the lateral ventricle. The patient lies with the face down, the neck flexed and head inclined to the side where the needle enters, and the replacement with air is done slowly. The roentgen-ray technic is as important and should be as painstaking as the operative procedure. The essential features are the position of the head on the plate and the angle of projection of the central ray.

As to the interpretation of Dr. Bassoe's pictures, I would say that the first case should have shown dilated ventricles, and the second an absolute cutting off of the ventricle at its center or in the region of the descending horn, the anterior and posterior horns showing up. In large tumors there are secondary manifestations resulting from edema, and this must also be considered in the interpretation of ventriculograms.

I believe, as Dr. Bassoe does, that the death of the second patient was due to acute increased intracranial pressure; and if this diagnosis could have been made early enough, the pressure might have been reduced by putting in a needle and allowing some of the air to escape, with the result that the patient's life might have been saved.

In regard to the indications for ventriculography: It can be utilized when tumors cannot be localized; in cases in which the patient has been operated on and the tumor not found; in cases of tumor in which the secondary symptoms are so pronounced as to overshadow completely the early localizing symptoms and signs. With our present knowledge of ventriculography, a careful clinical history and examination of the patient are absolutely essential. Ventriculography should be used only as a corroborative procedure, when necessary, or as an aid when it is impossible to obtain a reliable history or satisfactory examination of the patient. Ventriculography is thus in the same position as all other laboratory methods of diagnosis. I want to emphasize my opinion that routine ventriculography should not be practiced.

I am interested at present in determining how small a tumor may be and how favorably it must be located to be revealed by ventriculograms. In a young boy, a calcified tumor about $1\frac{1}{4}$ inches (3.17 cm.) in diameter, located in the frontal lobe, produced a definite displacement backward of the anterior horn of the ventricle visible in the ventriculogram.

The interpretation of ventriculograms in practice depends on a careful comparison of the ventricles of the two sides. Again, if ventriculography is negative in a case of suspected brain tumor, it should be repeated after a given time if the symptoms continue, just as is done when roentgenograms are made of other parts of the body. One such procedure does not negative the evidence of a tumor growth.

We all agree that any diagnostic method or procedure should be as safe as it is possible to make it. Only by reporting cases and the results observed,

especially cases verified by operative procedure or postmortem findings, shall we be able to arrive at a definite conclusion as to the value of this procedure in its entirety.

DR. LOYAL E. DAVIS: As one reads the literature on ventriculography and hears reports on this method of brain tumor localization, one is impressed with the total lack of consideration of the normal ventricle. Before any method which purports to recognize a pathologic variation in any organ can be placed in its property category, the normal appearance of that structure must become well established.

Retzius, many years ago, studied the lateral ventricles by constructing paraffin casts and called attention to a fact which has been lost sight of; that is, that about one third of all brains have a rudimentary posterior or occipital horn. Only recently, I had occasion to study the lateral ventricles in brains which were hardened *in situ* and then carefully suspended after removal. The ventricles were injected with mercury under fluoroscopic control, and roentgen-ray pictures were taken by Dr. Arthur Metz in an accurately lateral plane, all necessary precautions being taken to obviate errors in technic. Illustrations which I have show that numerous brains have blunted posterior horns instead of the long graceful extremity we are led to believe is normal. The striking variations in the shape and general contour of the body of the ventricle and the angle at which the inferior horn arises can be seen. It is quite obvious that such anatomic variations might lead to serious diagnostic errors. All of the brains so examined were later dissected, and any reason for such variations sought. In twenty-five per cent. of the brains so examined there were marked variations, particularly in the posterior horn.

In thirty-five cases of brain tumor in the service of Dr. Allen B. Kanavel during the last two years, ventriculography has been employed on two occasions. In one case, the right inferior horn was found to be deformed, but before operation quantitative perimetry revealed quadrantic hemianopsia. This fact is a real criticism of our haste in using the method. In the other case, a frontal lobe tumor, the amount of air injected was not sufficient to fill the ventricles. We have observed three rules in using this method: (1) not to change the position of the head after beginning the operation; (2) to withdraw fluid slowly and inject the air more slowly, and (3) to replace air equal in amount to three fourths of the fluid withdrawn.

DR. GEORGE L. DAVENPORT: In regard to the anatomic variations, it is true that the posterior horn presents the greatest amount of variation found. In the pictures of ventricles containing mercury, it appears that the brains had been removed from the skull before the mercury was injected. The occipital lobes rest on the tentorium and thus the posterior horns will be more flattened and pointed. I agree that variations in the size and shape of the ventricles must be taken into consideration and, as stated before, only by comparing the two sides, as in other roentgen-ray work, can a diagnosis be reached.

DR. LEWIS J. POLLOCK: The brains in Dr. Davis' cases were hardened *in situ*, an accepted method for the study of lateral ventricles, and roentgenograms were made by Dr. Arthur Metz, whose technic is beyond reproach. I think the findings may be accepted as fairly accurate.

DR. PETER BASSOE: I am glad that I brought up these two wretched cases, as I think the discussion will serve to clear up the subject. I was particularly glad to hear Dr. Davenport say that a careful neurologic examination should be made first in all cases, and that the air injection should be used only in

selected cases. This helps to counteract the unfortunate, but current, misunderstanding created by Dandy's paper in *The Journal of the American Medical Association* last year, to the effect that all brain tumors can be located by this method, and that a thorough neurologic examination is ~~not~~ necessary. It is also of practical importance to bear in mind the variations in the shape of the ventricles brought out by Dr. Loyal Davis.

CHRONIC TROPHEDEMA (MILROY-MEIGE DISEASE); A REPORT OF TWO CASES. DR. WILLIAM H. HOLMES.

On June 1, 1892, Dr. W. F. Milroy, Professor of Clinical Medicine and Hygiene in the Omaha Medical College, published an article entitled, "An Undescribed Variety of Hereditary Oedema" (*New York M. J.*, Nov. 5, 1922). It dealt with the case of a clergyman who presented persistent edema of the lower extremities. The family history of this patient showed that in six generations, comprising ninety-seven persons, there were twenty-two cases of this deformity. In five cases, both lower extremities were involved; in sixteen cases, only one lower extremity was affected; but, unfortunately, no mention is made as to whether the edema was on the right or left side. In one case, no mention is made as to whether the edema was unilateral or bilateral. One member of the group had an enlarged foot until maturity, at which time the testicles began to enlarge. As the enlargement of the testicles increased, that of the foot receded, until it was again of normal size. Although this case was not personally observed by Milroy, I mention the point because it is the only reference I have found in which improvement has been reported after the condition had become established.

Some seven years after the observation of Milroy, Henry Meige reported a number of cases and gave to the disease the name of chronic trophedema, by which designation it is still known. In this country, it is occasionally referred to as Milroy's disease, but in continental Europe the priority of Milroy's contribution is frequently overlooked. The term trophedema, without the use of qualifying adjectives, has been employed by Meige to designate all forms of dystrophic edema, the causes of which are unknown, but which appear to be due to some disturbance of the nervous system.

The disease is characterized clinically by edema which is so firm that the area involved does not pit on pressure. The overlying skin is usually glossy white, and may feel slightly cooler than that of other parts. There is no other evidence of trophic disturbance, such as ulcers, bony deformity, etc. The veins and lymphatics of the extremity affected are not visible. Although the disease may result in enormous enlargement of an extremity, joint motion is not restricted. Patients do not complain of pain, heat, etc., but they are constantly conscious of the increased weight of the affected part. Motor, sensory and reflex functions are normal. When the disease is established, it appears to persist throughout life, irrespective of treatment. Meige says that it persists without noticeable prejudice to the general health, but other observers have reported cases in which there seemed to be increased susceptibility to erysipelas infection of the edematous part. The disease may be congenital; sometimes it is congenital and hereditary, as in Milroy's cases; and it is also observed in adolescents whose family history is negative. It occurs more frequently in the female than in the male. In reading case reports, it has seemed to me that the left lower extremity is involved more frequently than the right. The entire extremity is not necessarily edematous, but only a segment. In order of frequency, then, the disease occurs in a segment or an entire lower extremity,

both lower extremities, one upper extremity, both upper extremities, face and thorax. It is frequently confounded with elephantiasis and with myxedema.

CASE 1.—Mrs. J. P., a white woman of American parentage, aged 42 years, first noticed an increase in the size of the right lower extremity about twenty years ago. Shortly thereafter a similar enlargement of the left lower extremity was noted. The swelling gradually increased until at the present time the ankles are 18 inches (45.72 cm.) in circumference. The swelling was not associated with or preceded by any subjective sensations other than a disinclination to move about because of the great weight of the limbs. For many years the condition has remained unchanged. She has only one other symptom, namely, great difficulty in staying awake. Her personal history is negative. She does not recall any serious illness, but assumes that she had all the ordinary diseases of infancy. Menstruation began at the age of 13. It has always been irregular, lasts for three or four days, and on the first day she has some pain. She was married at the age of 28, six years after her legs had become enlarged. Although no contraceptive measures have been used, she has never been pregnant. Her normal weight is 140 pounds (63.5 kg.), but recently she has gained about 8 pounds (226.8 gm.). None of the other members of her family are affected.

On physical examination, the negative findings to which I attached importance in arriving at a diagnosis were: a normal red cell count, white cell count and hemoglobin. A negative blood Wassermann reaction was obtained with two different antigens. The urine was normal. Results of examination of the heart and vessels, of the lungs, and of the abdomen and pelvis were negative. There was no adenopathy. The positive findings were: a hard edema of the lower extremities of twenty years' duration; dry, harsh and scanty hair; a small hard nodular thyroid gland; a retroverted infantile type of uterus; an inequality in the size of the pupils (left larger) with otherwise normal eye reflexes; a lack of adequate emotional reaction, with retarded intellectual processes. Neurologic examination was otherwise negative. The patient would not consent to the making of a photographic record, nor would she enter the hospital so that her metabolic rate could be determined. In arriving at a diagnosis, I considered: (1) the possibility of an obstruction of the lymphatic vessels, (2) an obstruction to the venous return, (3) a metabolic disturbance leading to edema. In regard to the first condition, there was no history of a lymphangitis; the lymphatic glands were not enlarged; a previous filarial infection seemed improbable; and further, the skin did not show the characteristic pigmentation, thickening and falling in folds over the ankle, which is seen in long continued lymphatic obstruction. If the enlargement had been due to venous obstruction there would have been cyanosis, coldness of the extremities, varicosities and a discoverable cause. A cardiorenal basis was excluded by the absence of cardiac signs, urinary changes, hepatic enlargement, etc. I, therefore, came to the conclusion that the condition was based on a disturbance of metabolism due to dysfunction of some of the internal secretory glands. The patient has, for more than a year, received various combinations of thyroid, suprarenal, pituitary and ovarian medication, without improvement.

CASE 2.—Miss E. D., aged 30, born in the United States of Scandinavian parents, was examined in Wesley Memorial Hospital, through the kindness of Dr. W. E. Schroeder. The family history was negative. Her personal history was negative up to the age of 15 years, at which time swelling of the left foot was accidentally discovered while having shoes fitted. The enlargement of the foot and leg gradually increased and attained its present dimensions

after the lapse of about eighteen months. She had never experienced any pain. There had been no infections or injuries. She had been admitted to the hospital four times. On her first admission, the blood was examined on three successive nights for filaria, but none were found. As no explanation of the edema could be found, a diagnosis of pseudo-elephantiasis was made. Another surgeon, in an attempt to relieve the edema, buried a strand of silk in the subcutaneous tissues on both the inner and outer surfaces of the leg, extending from the inguinal region to the ankle. She was confined to bed for three weeks, with the foot elevated, during which time the edema almost entirely disappeared. Following her discharge from the hospital, the edema returned almost immediately. Although this woman is 30 years old, she has never menstruated. Her face is infantile in appearance and is covered with a fine downy growth of hair. The hair of the scalp is harsh, brittle and scanty. The bodily development is that of a young boy. The breasts are not developed. There is no axillary hair and little pubic hair. She appears stupid and disinterested.

Examination revealed normal eyes, ears, nose, throat, heart, vessels, lungs and abdomen. Rectal examination revealed no masses in the pelvis. The uterus was small. There was no adenopathy. Blood counts and blood Wassermann tests were negative. Nonprotein nitrogen, blood urea and blood creatinin were within normal limits. Her basal metabolic rate was plus 9. The urine was also negative. The left leg appeared to be about twice the size of the right. There was a white, firm, painless edema which did not pit on pressure, extending from the ankle to the inguinal region. The edema was mostly above the ankle, the foot being only slightly edematous. Roentgenograms of the pelvis and extremities showed no changes in the bony structures. Motion, sensation and reflexes were normal.

Measurements in Cm.,			
Circumference	Right	Left	Difference
At the gluteal fold.....	47.0	53.5	6.5
Mid-thigh	44.5	49.0	4.5
Knee	34.5	45.0	9.5
Calf	29.5	45.0	14.5
Ankle	19.5	26.5	7.0

THE IMPRESSIONS OF NEUROLOGY IN RUSSIA AND GERMANY.

DR. GEORGE B. HASSIN.

During my recent stay of three months in Russia and Germany, I had the opportunity of meeting a great many interesting people. Among them were scientists, teachers, laboratory workers, clinicians and even communists, commonly known as bolsheviks. I also had the opportunity of visiting laboratories, hospitals and clinical institutions, as well as private homes of university professors, school teachers and physicians. From numerous conversations and personal observations, I gathered some information as to the political and economic conditions of Russia, as to the condition of the medical sciences in general and that of neurology in particular.

This report will be limited to a brief outline of the conditions in Russia where I spent nine weeks visiting various cities (Petrograd, Moscow, Kiev, Odessa, Nijni Novgorod, Kherson).

The neurologic work, the clinical part of it, is being carried on in Russia as usual. It never ceased. Even during the frightful years of the civil wars

that, following the bolshevik revolution, brought on terrible misery and poverty, the medical men, including neurologists, continued their hospital and clinical work. Because of terrible surroundings, lack of intelligent help, food, light, heat, animals and chemicals, research work suffered to an extreme degree. Yet, in some instances it was done as scientifically and thoroughly as in the best hospitals of this country.

The clinical material in Russian hospitals, especially in the wards and in the university clinics is very rich. The wards are frequently overcrowded. But the patients suffer, not so much from lack of medical skill, attention or nursing, as from need of medicines, food, and in winter, from the terrible cold. In cold weather they try to keep the patients more or less comfortable by piles of rags, old worn out overcoats and pillows, while the physicians make their rounds in overcoats, felt or rubber shoes and of course with their hats on. Even in the month of September, when the weather in Russia was comparatively mild, an attending neurologist was making rounds with his military overcoat on. This became, he remarked, a habit with them, not only in the hospitals, but also in government civil institutions. I was told that owing to the terribly cold winters, the absence of hot water, few habitable houses, overcrowding (whole families had to live in one room), poor sanitation or rather absence of it, lack of soap and warmth, undressing was out of the question. It was therefore impossible to keep one's body clean, and the result was horrible infestation of the hospitals and private dwellings with lice. In short, the hospitals were not only cold, deprived of light and warmth, but also exceedingly filthy. At the time I visited them, the conditions in some cities (Petrograd, Moscow, Kherson), thanks to the mild weather, were much better.

The clinical material, as I said, is very rich and of great variety. Neurosyphilis, postencephalitic states and brain lesions caused by typhus fever are especially abundant. Typhus fever is exceedingly common, owing to the terrible sanitary conditions alluded to. In winter, typhus fever is pandemic, affecting hundreds of thousands of people. For instance, in 1919, 650,000 cases were registered in Russia, of which 55,000 occurred in Petrograd alone, with a population at that time of 700,000. The death rate was about 10 per cent. It is no wonder, therefore, that this disease became a favorite object of study, numerous monographs and magazine articles having been devoted to various phases of this morbid condition, including the nervous system. Dr. Davidovsky of Moscow, in 1920, published a large work on typhus fever, in two volumes. The first volume of 350 pages has forty pages devoted to the nervous system. Though the paper is poor, the book has a number of good photomicrographs and beautifully colored illustrations, while the title page bears the famous slogan of the communists: "Proletarians of all countries be united." Davidovsky's studies pertaining to the nervous system, based on seventy cases, were carried on in the pathologic laboratory of the first state university of Moscow. They are of great scientific value and considered classic by his countrymen. However, there are a number of minor contributions on the same subject which, in my opinion, are not inferior. I would mention those by Anichkoff of Petrograd, Tiesenhausen and Jguenti of Odessa and Piette of Kharkoff. The specimens I had the opportunity to examine show practically the same changes seen in lethargic encephalitis. Except for the so-called nodules so abundant and constant in brains of typhus patients, there is practically no difference between these two morbid conditions, though it is claimed that in typhus the principle seat of the lesions is not so much in the midbrain, which is the case

in lethargic encephalitis, as in the medulla oblongata. More interesting is the statement that in every case of typhus fever the nervous phenomena predominate, however mild the case may be, and that encephalitis is always present.

Equally great are the opportunities in Russia for studying encephalitic changes in pernicious malaria, which is raging in the eastern provinces of the soviets, such as Ufa and Kazan, and also in some southern regions (Caucasus).

Much less favorable were the opportunities for studying epidemic encephalitis and its sequelae, apparently because for a long time the Russian neurologists did not know of the existence of this disease. Thus the medical association of the city of Kharkov, the capital of Ukraine, was taken rather by surprise when at a September meeting in 1920, Professor Zlatogoroff of Petrograd announced that the Germans had discovered a new disease known as sleeping sickness and that Noguchi had discovered the cause of yellow fever. The first intelligent report on epidemic encephalitis was made in April, 1921, by Dr. I. J. Tarashevitz to the society of neurologists and psychiatrists in Moscow. However, even before Zlatogoroff and Tarashevitz made their reports, cases of epidemic encephalitis in Russia were by no means rare; but they used to pass as atypical cases of typhus or typhoid fever.

The numerous cases of postencephalitic states, so-called pallidal and striatal syndromes, which I saw in Petrograd, Moscow and Kherson, invariably gave a typical history of lethargic encephalitis, and there can be no doubt that in addition to the epidemics of typhus fever, pernicious malaria, recurrent fever and typhoid, there was also one of epidemic encephalitis. At the time of my stay in Russia, this disease was well known and understood. One Moscow neurologist was rather proud of having seen seventeen cases. He was so impressed by his experience that he thought he was justified in writing a book on this disease. When I told him that the literature on epidemic encephalitis in western Europe and the United States was superabundant, that we have excellent monographs by Economo, Hall, Tilney and Howe, he replied that this did not matter. Neurology in Russia, he added, was German, and as the German book on encephalitis by Oppenheim and Cassirer was obsolete, a new book was urgently called for, and he was going to supply the demand.

There is no doubt that many books, pamphlets and magazine articles are being published in Russia, not for the sake of promoting science, but for other purposes. I was assured by competent and reliable men that the present rulers of Russia are trying to promote research. The more one publishes the more chances one has for obtaining food rations, and if a scholar has no contributions to his credit, he is doomed to starvation. Others, again, must show their literary activity in order to be promoted to a professorship, directorship of some institution or to a higher medical degree. In short, the scientific spirit is at least somewhat, and probably very much, darkened by purely materialistic aspects.

The splendid neurologic periodicals of Bechterew, Popoff and the so-called Korsakoff journal of neurology are no more. Small sporadic volumes containing articles on neurologic subjects are being published but very irregularly, some good contributions appearing also in larger general medical monthlies. But special neurologic journals such as we have in this country, Germany, France, England and Italy do not exist in Russia. In fact, serious scientific workers in the field of neurology who would be capable of keeping up a high grade periodical abstain from publishing anything, preferring to do something

else to save themselves from starvation. For instance, a celebrated histopathologist held at one time about four government positions on which he had to spend the whole day, as he had to cover great distances on foot. In four years he has not published a line, on account of lack of animals, chemicals, proper temperature in the laboratory and absence of the most necessary facilities. Yet he is interested in many problems which he is quite capable of solving. For instance, he found that glycogen normally present in the lower portion of the spinal cord in birds is greatly increased when they are fed with rice. His specimens show the presence of this substance, not only in the spinal cord, but also in the subarachnoid space, where this catabolic product is discharged or, as the Germans put it, transported. The study of this interesting problem had to be discontinued; for, as he assured me, though he could obtain rice, he has no means with which to buy hens or pigeons. Before the revolution his family was wealthy. Even if animals could be obtained, he said, they would either be stolen or killed. There are many other problems, such as improving on some staining methods much used in neuropathology, which this excellent investigator had to abandon for lack of proper surroundings. In fact, as he justly remarked, how can one methodically and successfully do research work, especially on a large scale, when the laboratories are bitterly cold or damp, when the limbs are almost frozen, swollen or cracked, when one is underfed, physically exhausted and normally depressed? It is quite true that some exceptionally vigorous persons were doing brilliant work even under such frightful conditions. Thus it is related that the famous surgeon Veliaminoff, although literally starving and freezing (he had to hunt for a warm place), succeeded in completing important books, such as "Surgery of the Joints" and "Military Field Surgery"; and at the time of death he was writing a textbook. He died, they say with a pen in his hand. This occurred at the time of the bolshevik reign of terrorism, when the intellectual class was practically doomed.

To the horrors of starvation, cold, insufficient clothing and filthy surroundings should be added absence of foreign medical literature. During the time of blockade, when letters, books and parcels of any kind were being refused to Russia, no literature could reach that unhappy country. Even at the present time, when it is possible to obtain from abroad what some over there call "mental food," the great majority of scientists are deprived of the means to do so. The common cry is: "Give us mental food" in the form of books, reprints, periodicals, pamphlets or monographs. They can offer nothing in exchange, except rich pathologic material which they possess in abundance, but which unfortunately can be utilized very little. Neither can they preserve it for the future, for lack of containers and necessary preservatives.

The priceless material from typhus fever, starvation and various psychoses is either only partially utilized or has to be thrown away. Especially is it to be regretted that brains from persons who have died from starvation had to be destroyed. The starvation problems have been studied clinically, statistically and physiologically; even the general pathology was covered. Three volumes on starvation have been published by the health commissariat of the city of Moscow. A book on so-called epidemic dropsy or "Oedem-Krankheit" of the Germans has been published by Dr. Luria of Kazan. It contains good descriptions of the changes in the muscles and some ductless glands (thyroid, parathyroid and testicles), but nothing on the condition of the nervous system.

This he informed me he did not study because he did not have the necessary facilities. Nor did anybody else study the condition of the central nervous system in starvation, a problem of great interest. Unfortunately, they expect another famine this winter, especially in southern Russia, and there will thus be ample opportunities for such studies.

Starvation brought forth a dreadful psychotic phenomenon—cannibalism. Cases of the latter undoubtedly occurred, not only in the Volga regions, but even in the more civilized southern towns, such as Kherson. Some wretches would be satisfied by eating the flesh of children they previously killed, while others would sell it openly in the form of sausages and cutlets. Some of the former group of cannibals would ultimately commit suicide, while those of the latter variety were often found to be insane. Even more terrible, unbelievable, tales have been related; as, for instance, the using of the flesh of dead bodies for eating, feeding and commercial purposes.

Another strange psychotic phenomenon observed was the complete indifference of the population to the unfortunate sufferers from hunger. Many grown persons, and especially children, were literally dying in the streets, filling the air with moans, groans, cries and pitiful begging for bread. One physician told me it was not possible to concentrate one's mind on any kind of work. Yet one would pass by the dying victims of incredible sufferings paying no attention to them. In six months, there perished from hunger about 13,000 persons in the city of Kherson, which had a normal population of 89,000. In many cases, not a single member of even large families survived, seventy such families having been found dead in one section of that town. The sensorium I was told became so dulled that one was incapable of comprehending or in any way responding to so great a calamity. There undoubtedly are, in present-day Russia, many social and purely scientific problems of great value and interest to a neuropsychiatrist. The entire convulsive catastrophic movement known as the Russian proletarian revolution possesses many traits of a mass psychosis, for which the nervous system of the future generations of Russia will pay dearly. It is a pity that many innocent people and among them excellent men of science had to perish. Some did succeed in escaping death, but living as they do among pathologic surroundings, they are able to utilize only partially the rich material which is of so incalculable value to science. It is also pitiful to witness the gradual though slow decay of the magnificent clinical buildings, veritable palaces, in Petrograd, Moscow and other places, for there are no means with which to keep these places in proper condition.

The impressions are thus exceedingly sad. In fact, the entire state of affairs in the land of the soviets—the industries, common education, higher education, commerce and trade, science, the housing question, etc.—in short, the entire life, is a profound tragedy and misery.

Comparatively better is the situation in Germany. The atmosphere there is not so depressing as in Russia, and though much work is being done in the laboratories and hospitals, it is greatly hampered by the necessity of giving much valuable time to the struggle for existence. On account of the uncertain future, the necessity to think of daily bread, and lack of proper means, the scientists of Germany do not feel happy, they are much depressed, yet they continue in their work; and, like our Russian brethren, they are much to be admired, but still more to be pitied.

NOTES ON NEUROLOGY AND PSYCHIATRY IN AUSTRIA AND
• GERMANY. DR. N. L. BLITZSTEN.

During my recent stay of five months in Vienna, I was especially interested in the psychoanalytic movement which has had a rapid growth during and following the war. At present, there are in Vienna about fifty analysts in the Freudian entourage. About 30 per cent. of these are laymen, and a large percentage of the others are physicians without previous training in neurology and psychiatry. The Psychoanalytic Society of Vienna is composed entirely of Freud and his disciples. At the meetings, which are held about once a month, the dominating influence of Freud is felt at all times. There is seldom, if ever, any sidestepping from Freud's original postulates, merely further enlargement of them. Practically all of the psychoanalytic instruction is in the hands of the Freudian group. During the time I was in Vienna, there were no courses in psychoanalysis given at the university; and only one psychiatrist, Paul Schilder, was giving occasional psychoanalytic demonstrations in the Wagner-Juaregg clinic. The method of instruction is based primarily on individual analysis of the student, and secondarily, on a series of lectures on theory by members of the Freudian group for those students who are being analyzed. In addition, several of the older analysts devote one or two evenings a month to a discussion, for younger analysts, of analytic problems. In Berlin, conditions were similar. Lectures were given in the Psychoanalytic Polyclinic by Karl Abraham and Hans Sachs. Here the younger analysts have the opportunity to carry on their analyses under the direction of the older men.

The duration of a Freudian analysis is about six months, although this is decided during the course of analysis and depends on the overcoming of resistance. No cure is promised, and the patient is told that if the analysis ends unfavorably, the analyst is not to be held responsible. A certain degree of sexual abstinence is asked of the patient. No important decision is made during the analysis. The analyst must not show too great an interest in the cure and should never go beyond the limits of therapeutic interest. The important factors in an analysis are the overcoming of the resistance and the handling of the transference, which is in the end destroyed. It is especially important to know when to analyze the transference. Suggestions of any kind should not be made if the transference is to be handled successfully. The chief work is the affect work. In the analysis, the patient lives through all of those incidents which became pathologic in childhood. The analyst becomes the representation of those figures important in the life of the analysant. At the end of the analysis, the affect neurosis becomes a transference neurosis, which, in turn, disappears with the successful analysis of the transference.

In Vienna, as elsewhere in Europe, there is a strong reaction against psychoanalysis. This is due not only to the natural hostility of the conservative minded to the extreme tenets of the Freudian school, but also to the fact that an ever increasing number of laymen and physicians untrained in neuropsychiatry are practicing this specialty. Incidentally, I might mention that hypnosis for investigation and therapy is increasing in popularity in many of the larger neuropsychiatric clinics.

Owing to economic conditions resulting from the war, the progress of neuropathologic research has been greatly retarded. Valiant efforts to maintain previous high standards in this field have been rendered almost futile in some instances because of the lack of funds for even ordinary equipment.

Few are able to devote much time to research because of economic pressure. Many have assumed an attitude of extreme hopelessness because of chaotic conditions.

Clinical neurology has made greater progress, and the clinics of Austria and Germany offer a wealth of material. Especially numerous are cases of neurosyphilis and postencephalitic states. In spite of the ever increasing burden of upkeep, the hospitals are full to overflowing.

Especially interesting in the Wagner-Juaregg clinic in Vienna is the treatment of general paresis and tabes by subcutaneous injection of tertian malarial parasites. Many favorable results are reported, especially when the treatment has been given early in the disease. I had occasion to examine a small group of patients shortly before and after the treatment was given, and I was greatly impressed by the marked and rapid improvement. Schacherel, in the same clinic, uses some form of fever therapy in all forms of neurosyphilis, in addition to the usual antisyphilitic measures. He obtains his best results with typhoid and staphylococcic vaccines injected intravenously. Comparatively little intraspinal medication for syphilis is used in most of the clinics of Europe.

Redlich has been experimenting with inhalations of chlorylen (trichloroethylene) in the treatment of trifacial neuralgia. In many cases complete relief of pain is obtained, especially when the malady has existed for a short time and only one branch of the nerve is involved. Several of the men in Vienna are treating myoclonias and parkinsonian syndromes following epidemic encephalitis with intravenous injection of the patient's own spinal fluid, and they are obtaining favorable results in a large proportion of cases.

In Berlin, where I spent four months, Krause and his assistants are doing considerable experimental work on the autonomic nervous system. They have shown that there is a passage of calcium and potassium from the circulating blood in any stimulation or paralysis of the vegetative system. An isolated stimulation of the sympathetic or parasympathetic nerves by toxins which act peripherally is impossible because of the activity of a regulating mechanism located in the subthalamic centers. Assuming that the proportion of potassium and calcium ions is an expression of the functional conditions of the cells innervated by the parasympathetic and sympathetic nerves, the electrolytic balance must change in favor of one or the other ion in vagotonia and sympathicotonia. Preponderance of potassium corresponds to parasympathetic stimuli; preponderance of calcium, to sympathetic stimuli.

F. H. Loewy is trying to demonstrate a functional relation between the corpus striatum and the liver. In animals whose livers had been damaged by metallic salts, such as manganese, or by hemolytic serums, he found decomposition ferments in the serum against liver and brain, as well as degenerative changes in the corpus striatum.

In the treatment of epilepsy most of the clinics in Germany employ phenobarbital and bromids in combination rather than separately. Peretz believes that many cases of idiopathic epilepsy are of spasmophilic nature. He treats all these patients with intravenous injection of calcium chlorate, in addition to the usual medication, thereby obtaining, he believes, a larger proportion of cures. Peretz also has found a decrease in the lipid substances in the serum of syphilitic persons, especially lecithin. He believes that many symptoms of tabes and other forms of neurosyphilis are the result of a disturbed function of the nervous system, which is caused by the chemical combination of the syphilitic toxin with the lipoids of the nervous system. By introducing lipoids into the system, this chemical process can be retarded or stopped, and those

symptoms which are not due to actual degeneration of the nervous system altered. On this basis, he increases the lipoid content of the diet of his neurosyphilitic patients in the form of butter, eggs, cod liver oil and lecithin. This, in addition to the usual antisyphilitic treatment, results, he claims, in a much more favorable outcome in all forms of neurosyphilis, especially tabes.

In the field of psychiatry and the psychoneuroses, there is a general tendency throughout Germany and Austria to break away from the old classification of disease units. As a result, a group of phenomenologists has arisen who make intensive psychologic studies of individual symptoms. The tendency is to group psychologic and biologic types that merge into each other and show evidence of unusual biologic and phylogenetic tendencies which are preformed in the psyche and are brought out in the psychosis but not caused by it.

Intelligence testing among psychiatrists has made little headway in either Germany or Austria. There are no standard performance tests or general psychometric scales in use by psychiatrists. The mental status is diagnosed by individual judgment based on personal clinical experience. The work in mental tests by German psychologists apparently has not penetrated the domain of child psychiatry. The work of America in this, as in other fields, is practically unknown in Germany. This may be due to the prohibitive rates of American periodicals at the present exchange.

The clinics of Germany and Austria still have a wealth of material, and although few Americans are taking advantage of the opportunity, they still attract a large body of other foreign students. Despite the general anti-American feeling throughout Germany and Austria, I saw no evidence of hostility toward American physicians in any of the clinics which I visited.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 24, 1922

C. H. FRAZIER, M.D., *President*

A CASE OF SYPHILITIC HEMIPLEGIA WITH UNUSUAL TROPHIC DISTURBANCES. DR. C. A. PATTEN.

A case of hemiplegia was presented with trophic disturbances, consisting of marked destruction of bone and of bedsores, limited to the paralyzed side. There were probably two factors responsible for this condition: (1) the intense syphilitic disease, and (2) the sensory loss.

A colored man, over 60 years of age, was admitted to the Philadelphia General Hospital, July 28, 1922. He had had a stroke five years before, from which he recovered; a second one occurred eight months before and was followed by a semiconscious state for two weeks.

The clinical findings on admission were: the usual manifestations of right hemiplegia with involvement of the lower half of the right side of the face; marked disturbance of all forms of sensation on the entire right side of the body, including the sensory division of the fifth cranial nerve; decubiti on the shoulder, sacrum, hip, ankle and leg of the right side of the body; marked edema of the right upper and lower extremities. Incontinence of urine had persisted since admission. On the left side, the reflexes were prompt and sensation was normal. Argyll Robertson pupils, tremors of the face and left arm, motor

aphasia and mental deterioration were also present. The blood and spinal fluid gave strongly positive reactions for syphilis in all tests. Roentgen-ray examination revealed the following: "Inner surface of the head of the humerus on the right has a ragged outline, the posterior border of the glenoid cavity is not demonstrable, with bony proliferation about this area. Roughening of the outer border of the external condyle of the right elbow, bony proliferation of the great trochanter on the right, and slight erosion of the outer condyle of the femur are present. There is a suggestion of thickened periosteum of the inner upper one-third of the tibia. No pathology of any of the bony structures of the left side of the body or of the skull can be demonstrated."

Under proper care the decubiti began to improve, but the improvement was more rapid under small doses of neo-arsphenamin. At the present time there is only one unhealed lesion, which is on the leg. The incontinence has decreased, and sensation is less involved on the right half of the body.

The hemiplegia is undoubtedly on a syphilitic basis, and the sensory loss on the right side of the body predisposes to decubiti. The presence of an intense systemic syphilitic infection, the loss of mobility of the right side and sensory changes probably account for the marked bony changes.

DISCUSSION

DR. ALFRED GORDON: In the psychiatric department of the Philadelphia General Hospital at the present time is a patient who has shown some mental phenomena suggestive of paranoia and whose history is exceedingly interesting from the point which Dr. Patten has introduced. Before he entered the Philadelphia Hospital, the patient had been in another institution for cerebrospinal syphilis. He presented a deplorable condition—involvement of the sphincters, eyes and upper extremities. The condition progressed, and he was unable to move any of his limbs. He began to develop progressive muscular atrophy of the Aran-Duchenne type, later with involvement of the muscles of the shoulders. Energetic doses of neo-arsphenamin brought about improvement, and the patient was able to walk, the muscles of the hand began to fill out, and at the end of eight or nine months the condition of the sphincters improved. I saw him at the Philadelphia Hospital recently; he was able to walk, had full use of his limbs, and had no disturbance of the sphincters. The most remarkable feature was the disappearance of the muscular atrophy.

DR. CHARLES K. MILLS: This case is interesting in connection with the question of the possible existence of special trophic centers in the cerebral cortex. We are all familiar with many cases of hemiplegia in which there is little atrophy, although there may seem to be wasting from disuse, and we are also familiar with many cases of hemianesthesia of cerebral origin in which little or no trophic changes are present in bone or muscle, or even in the skin or subcutaneous tissues. Many years ago, Savill and another English medical writer reported cases which they believed indicated that there were trophic centers independent of sensory and motor centers proper. I have seen some remarkable cases of trophic disorder in hemiplegic cases, one in particular in which extraordinary atrophy was evident on the side of the hemiplegia.

A CASE OF PARAMYOTONIA. DR. ALFRED GORDON.

The classical myotonia of which Thomsen's disease is an example presents some variations as to the onset, distribution, etiology and mechanical irritability. The chief characteristic symptoms are: sudden rigidity of the muscles

on attempt to use them, increased mechanical irritability and finally special electrical reactions. A galvanic current will produce a wavelike contraction of the muscles running from the cathode to the anode, also gradual relaxation. Each of these characteristics may vary in intensity. Eulenberg and others described a variety called paromyotonia, in which the muscular spasm occurs not on motion but on exposure to cold, and in which the electrical reaction with Erb's formula may be entirely absent. In other varieties of myotonia there has been a different distribution of the muscular spasms. Myotonic contractions of the muscles of the face have been mentioned by writers, but always in association with involvement of the body. The following case is an example of paramyotonia in which the spasm is confined exclusively to the muscles surrounding the orbits and especially to the eyelids:

A man, aged 52 years, after prolonged mental distress of a domestic character, developed a myotonic contraction of the muscles surrounding the base of the nose and of the orbicularis palpebrarum. Suddenly, without apparent cause, but often on the appearance of a bright light, the muscles around the orbits would contract intensely, the eyes would close tightly, and he would be unable to open them. The condition would persist for many minutes, and relaxation of the muscles would take place rapidly but not suddenly. The patient learned to shorten an attack by a certain turn of his head or by blowing his nose. Further examination, together with the past and family histories, revealed nothing abnormal. Urinalysis and the Wassermann test of the blood were negative.

After a period of treatment with intensive doses of strychnin, the spasm of all other muscles disappeared, but the tonic contractions of the eyelids remained. Mechanical irritability was increased. Testing with electrical currents, galvanic and faradic, gave a prompt response, but at no time was there the classical myotonic reaction of Erb.

The interesting points in this case are: absence of myotonic reactions and of any special etiologic factor immediately preceding an attack; the limitation of the spasm to the muscles of the eyelids which makes this case unique, since in the records of the literature the facial muscles were invariably involved, together with other muscles of the body.

DISCUSSION

DR. WILLIAM G. SPILLER: In myotonia the contraction is introduced by voluntary movement of the part. I have watched this man, and it is not a voluntary movement that produces the contraction of the lids, nor is the contraction confined to the lids. He has also a spasmodic contraction about the muscles of the mouth. I can see no reason why the condition could not be considered bilateral spasm of the facial nerve distribution.

DR. CHARLES K. MILLS: I never have seen a case of myotonia like this, if it is myotonia. Dr. Gordon failed to tell us of the refraction and the retinal condition. I have seen cases somewhat like this, which seemed to me to be rather a form of a neurotic or hysterical blepharospasm. It is well known that in certain irritable conditions of the eyes associated with a similar state of the nervous system blepharospasm or a facio-blepharospasm is not uncommon.

DR. ALFRED GORDON: I know that in blepharospasm the eyes close and open repeatedly and rapidly, but a condition in which the eyes are tightly closed for as long as ten minutes I do not know. In regard to hysteria, as Drs. Leopold and Mills suggested, the man was examined from every possible standpoint and

that condition was considered. His entire make-up, his previous family and personal history, the absence of hysterical manifestations, not only physical, but mental, absolutely speak against hysteria.

PARALYSIS OF THE UPWARD ASSOCIATED MOVEMENTS OF THE EYEBALLS. DR. WILLIAMS B. CADWALADER.

My object in presenting these cases is to call attention to the occurrence of paralysis of the upward associated movements of the eyeballs from totally different causes. In the first case, there was probably a hemorrhage implicating both oculomotor nuclei, caused by coughing. Similar cases have been presented before this society by Dr. de Schweinitz and Dr. Spiller.

A boy, 14 years old, was admitted to the University Hospital Oct. 25, 1922. In September, 1922, he developed bronchitis with severe attacks of coughing. Soon after an exceptionally severe paroxysm, he noticed diplopia.

On examination, in addition to the signs of general bronchitis, it was found that there was paralysis of the superior rectus muscle on each side. There was decided limitation of the upward associated movements of the eyeballs. There was no involvement of the ciliary muscles. The rest of the examination was negative, except that the patient complained of marked polyuria. This might be attributed to involvement of the tuber cinereum.

The second case was presented here by me and is recorded in the proceedings of the Society (*Arch. Neurol. & Psychiat.* 4:237 [Aug.] 1920); at that time the diagnosis was obscure because the paralysis of the associated movements was the only striking localizing sign. Since then, however (and it is for this reason that I now call attention to the case), the patient has developed marked disturbance of speech, scanning in character, marked tremor of the right upper and lower limbs, with increased tendon reflexes and ankle clonus on that side, and also intention tremor of the left upper limb. I believe this is a case of multiple sclerosis, implicating the oculomotor nuclei; the symptoms seem to be progressively becoming more pronounced.

DISCUSSION

DR. WILLIAM G. SPILLER: In the two papers which I have published on this subject (*J. Nerv. & Ment. Dis.*, 1905, and *Arbeiten aus dem Neurologischen Institut, Vienna*, 1907) I have given all the information I have been able to obtain. In the latter paper, I referred to nineteen necropsies in which a lesion near the aqueduct of Sylvius was found, and to this list I added another case.

I do not believe a lesion far from the oculomotor nucleus is capable of producing a persisting paralysis of upward or downward associated movement, as paralysis of associated movement of cortical origin is always transitory, depending on the bilateral innervation of the nuclei of ocular muscles. The lesion need not be in the corpora quadrigemina, but must be near enough to the oculomotor nuclei to affect the path from each cerebral hemisphere. The lesion may be so small as to escape detection unless a thorough microscopic examination is made. In the case I reported the tumor was so small it might easily have escaped detection. The fact that paralysis of upward or downward movements often is complicated later by paralysis of other ocular muscles also establishes the existence of a lesion near the nuclei.

DR. J. HENDRIE LLOYD: I once saw paralysis of the upward associated movements of the eyes in a case of pseudobulbar palsy. The man had a history of syphilis; he had had several slight hemiplegic attacks, and when he

came under my care he had complete paralysis of the tongue, the lip muscles and of the muscles of mastication and swallowing. Necropsy revealed lesions in the lenticular nuclei. These associated movements of the eyes are undoubtedly represented in the brain cortex; and any lesion that interrupts the connecting fibers between these cortical centers and the nuclei in the mid-brain may cause paralysis of associated movements. When this occurs in pseudobulbar palsy, it has been called a pseudo-ophthalmoplegia. The present case is probably not of that kind, but I mention the subject as showing that the lesion in palsy of the associated movements of the eyes is not necessarily in the midbrain nuclei.

DR. WALTER FREEMAN: It is true that we see paralysis of associated movements of the eyes in such cases of pseudobulbar palsy as Dr. Lloyd mentions, and the differentiation of this condition from the paralysis occurring as a result of interference with the condition below the center is of importance. Bielschowsky has suggested two methods for eliciting reflex movements of the eyes when voluntary movements are no longer possible: one is the use of the aural douche according to the method of Bárány, and the other consists in having the patient fix with his eyes an object directly in front of him, and then turn his head passively to either side. The eyes, fixed on the object, will rotate to the physiologic limit.

DR. CHARLES M. BYRNES: Through the kindness of Dr. Spiller, I have just examined the brain of a syphilitic patient who had paralysis of upward associated movement. There was no lesion of the corpora quadrigemina or nucleus of the third nerve, but there was definite infiltration and disintegration of both lenticular nuclei.

SINGULTUS CRISIS IN TABES DORSALIS. DR. C. M. BYRNES.

A case of singultus crisis in tabes which was perhaps related to a gastric crisis and in all likelihood induced by intradural therapy, is reported.

I could find only three instances in the literature of this symptom associated with tabes. Daunic reported the condition first, and several years later Stembo recorded two instances in which the affection was relieved by induced vomiting.

In my case, the attack developed shortly after the intradural administration of mercurialized serum and persisted continuously for a period of ten or eleven days. The hiccup continued during sleep and while the patient was under ether anesthesia. Various well-recognized methods of treatment were adopted without beneficial effect. Assuming that the singultus in this case was induced by the intradural treatment, it did not seem probable that the mechanism of the attack was dependent on direct phrenic or diaphragmatic irritation, but it did not seem likely that it was due to irritation of the thoracic nerve roots. While it is generally known that the diaphragm receives sensory and motor fibers from the last thoracic nerve, the recent studies of Felix have shown that at least the last six or seven thoracic nerves also enter into the innervation of this muscle.

In two cases of gastric crisis, I have succeeded in relieving the condition at once by means of intradural therapy, and I concluded, therefore, that in this case of singultus crisis it might be well to adopt this method of treatment. Hence, on the eleventh day of the patient's illness an intradural dose of mercurialized serum, containing $\frac{1}{50}$ grain (0.0013 gm.) of mercuric chlorid, was administered. The hiccup ceased immediately, but on the following day there

were several less vigorous and less rapid contractions of the diaphragm. These soon ceased, and the patient has had no further attacks after a period of ten months.

Lantern slides were then shown of the cutaneous anesthetic areas discovered on the thoracic wall, of the diaphragmatic innervation according to Felix and of a diagram constructed by myself to illustrate the intercostal and phrenic innervation of the diaphragm.

NATURE OF CERTAIN FUNCTIONAL NERVOUS DISTURBANCES
AND THEIR TREATMENT ALONG METABOLIC LINES. DR. RALPH
PEMBERTON.

This paper is published in full on page 208 of this issue.

DISCUSSION

DR. THEODORE H. WEISENBURG: In a number of arthritic patients who had migraine, improvement in the former disease caused a disappearance of the migraine. This emphasizes the fact that the same underlying causes may exist for arthritis as for symptoms occurring in the psychoneuroses.

In those patients in whom the removal of focal infection does not eliminate the arthritic symptoms, Dr. Pemberton has obtained successful results by limiting the food intake. There is no doubt that a similar method in the treatment of certain types of psychoneurosis will be of value. On the other hand, there are patients in whom overfeeding by the Weir Mitchell method accomplishes the best results.

The treatment of psychoneuroses is by no means a question of the removal of focal infection or underfeeding or overfeeding, but in large part it is a problem of internal medicine in conjunction with the aid of a well conducted biochemical laboratory.

DR. WILLIAM G. SPILLER: Dr. Pemberton's remarks have historical interest. Charcot some thirty years ago, in his "*Leçons du Mardi*," spoke of the relation of arthritic disorders to nervous diseases, especially the functional forms; and he emphasized the fact that nervous disorders frequently occur in families subject to arthritic defects. In one place, he says that arthritic manifestations, such as gout, articular rheumatism and cutaneous affections, form a tree; and nervous disorders, such as neurasthenia, hysteria and epilepsy form another tree. The two grow on the same soil and communicate by their roots, and their relation may be so close that one may suspect that they are of the same tree. Dr. Pemberton has given us the common origin in metabolism and biochemistry.

DR. PEMBERTON: I did not know Charcot had made the statements Dr. Spiller quotes. I knew that the relation had been recognized by many people, but I did not know that Charot had called attention to that syndrome.

Book Reviews

MENTAL DISEASES. A Public Health Problem. By JAMES V. MAY, M.D., Superintendent, Boston State Hospital. With a Preface by THOMAS W. SALMON, M. D., Professor of Psychiatry, Columbia University. Cloth. Price, \$5 net. Pp. 544. Boston: Richard G. Badger, 1922.

This is a book full of the statistics and the history of mental diseases. If one prepares to be disappointed on seeing the great number of quotations, one is pleasantly surprised. It is a compilation of the rare right kind.

The first part is developed from Dr. May's work on the Commission on Statistics of the American Psychiatric Association. It emphasizes the importance of mental diseases for every citizen—with an incidence of 566 per 100,000 of the population, an investment of public funds to the amount of \$187,000,000, and appropriation of \$33,500,000 each year, with a waste from patients out of hospitals which exceeds all these figures. The evolution of the modern psychiatric hospital is traced, with the laws and methods of administration which concern it. Such statements as the following are too often neglected, but will repay the most careful consideration. For the average state hospital reception buildings should be built to care for 6 per cent. of the population, convalescent buildings for 4 per cent., hospital for 2 per cent., infirmaries for 8 per cent., wards for disturbed cases for 20 per cent., for epileptic patients, 3 per cent., planning on quarters for working patients for 40 per cent., the quiet, clean, and appreciative chronic class 14 per cent., and the tuberculous 3 per cent. This and other statements about planning a new hospital are so important that it would be criminal for a commission having such a project in view not to have such sources of information as this book at hand for consultation. Chapters follow on the psychopathic hospital, mental hygiene, immigration, war psychiatry and criminal responsibility. There is a most interesting plea for a better use of the statistical method contained in a chapter on the modern progress of psychiatry.

In a sense, the second division of the book begins in two chapters of Part I on etiology and endocrinology, the former using history and statistics to make its points, and the latter built up entirely of others' opinions in chronologic order. In Part II a chapter is devoted to each of the twenty psychoses listed in the "Classification of the American Psychiatric Association." The method is not that of a textbook, but has an interest of its own. The first chapter, for instance, on "Traumatic Psychoses," refers to ancient writers, then to the crowbar case of 1848, Ericksen's "Railway Injuries" of 1866, the newer ideas brought forward later by Oppenheim and Charcot, and gives in longer abstract Meyer's important contribution in 1903. The extremely interesting "post-traumatic constitution" is well described. The subject is then covered according to Kraepelin and Bonhöffer, with excellent reviews of the character changes which often occur. A few words describe postmortem findings, widespread destruction of nerve cells, glial proliferation and capillary softenings. The chapter concludes typically by quoting the limitations given by the standard classification and a percentage of the admissions of patients with this diagnosis to forty-eight state hospitals (0.3 per cent.).

Other chapters follow the same form, and as the changing ideas that have been published during the centuries and decades are given, a picture of the particular disease is built up which has considerable vividness. There is no attempt to get a cross section of the disease, which would be satisfactory for textbook use. There is a certain looseness in the chapter on alcoholism. We note that in 1909-1912 there was an admission rate of 10 per cent. for alcoholic psychoses to New York State Hospitals, and this rate dropped to 1.9 per cent. in 1920, a prohibition year. When one considers the size of the illicit drug traffic, it is surprising that drug psychoses furnish only 0.39 per cent. of the admissions to state hospitals.

This book is a novel and stimulating one for the psychiatrist! It would be hard to forecast what its effect would be on any one else.

FUNCTIONAL NERVOUS DISORDERS: THEIR CLASSIFICATION AND TREATMENT. By DONALD E. CORE, M.D., M.R.C.P., Honorary Assistant Physician, the Manchester Royal Infirmary. Cloth. Price, \$6. Pp. 371, with 21 illustrations. New York: William Wood & Co., 1922.

The purpose of this book is to make a classification of functional disorders which shall be free from the "vagueness" which obtains at present. The foundation for this is sought in certain psychologic concepts, descriptive rather than explanatory, which are outlined dogmatically and in terms which often make reading wearisome. Case studies are conspicuously absent, though illustrative samples are occasionally introduced. Although avowedly dealing with behavior as such, the author insists on the absence of evidence of structural deficiency as a *sine qua non* for the diagnosis of functional disorder. Apparently, it makes no difference whether the structural defect is such as to determine the behavior in question. Any evidence of feeble-mindedness (alluded to as dementia!) or acquired dementia eliminates a diagnosis of functional disorder. Yet one may perhaps legitimately wonder what is the basis for the "defective control of the emotions" on which so much stress is laid. Apparently, "hysterical people, though varying among themselves in their intellectual powers, are as a class less intelligent . . . than those who are not so orientated"; and yet, if the grade of intelligence falls below the imaginary line which delimits normal intelligence, while the person "may show hysterical behavior," this cannot be considered as true hysteria but only as a symptom of the defect.

It is unfortunate that the author has been so ambitious, as there is much in his views that is worthy of serious consideration, but which at present seems to be poorly digested. In dealing with treatment, little use is made of the theoretical considerations which make up the bulk of the book, and the discussions are eminently practical. It seems, however, unfortunate to assert, without proof, that the so-called "mnemoneuroses," which form the "majority of cases of functional nervous disorders as met with in modern civil life," lead almost inevitably, if not remedied, to "absorption of pressor substances, and hyperpiesis—with its consequences, cerebral, cardiac and renal arteriosclerosis."

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THE DEEP SENSIBILITY OF THE FACE *

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Residual sensibility following various definite peripheral nerve lesions has afforded the material for many original and productive investigations. The contributions of Henry Head and his collaborators have marked an epoch in the study of the peripheral sensory mechanism, and the numerous nerve injuries encountered during the Great War gave added impetus to further experiments. By virtue of their comparative inaccessibility and the scarcity of verifiable clinical lesions, little is accurately known of the sensory distribution of the cranial nerves in man. This is quite true of the sensibility of the face, the cranial nerve supply of which must be the trigeminal or facial nerves or both.

Since Sir Charles Bell showed that facial palsy was dependent on a lesion of the seventh cranial nerve, for all practical purposes this nerve, with the exception of its chorda tympani branch, has been regarded as a motor nerve. The nervus intermedius without doubt carries these afferent fibers from the chorda tympani to the brain stem where they enter the tractus solitarius and course further cranialward. On the other hand, it has been commonly assumed that destruction of the trigeminal nerve results in the loss of all forms of sensation in the face and on the tongue, with the exception of taste. The perfection of the surgical technic in operations on the sensory root of the fifth cranial nerve for the relief of trigeminal neuralgia has afforded material for accurate clinical investigation of the sensibility of the face. On examination of the sensation of the face following trigeminal neurectomy, it was noted in a number of cases that deep pressure stimulation elicited a response, although all forms of superficial sensibility were lost. This led to the examination of all of the clinical cases for the absence or presence of deep sensibility and to the investigation of sensation following lesions produced in experimental animals.

* Contribution No. 103 from the Anatomical Laboratory, Northwestern University Medical School.

In studying the deep sensibility of the face, it has been my purpose to attempt to establish two points. Is pressure pain sensibility lost after division of the sensory root of the trigeminal nerve? If this type of sensation is not lost, do the fibers which transmit pressure pain travel cephalad in the facial nerve?

LITERATURE

In 1905, Henry Head¹ and his co-workers first called attention to the presence of fibers contained within the nerves supplying the muscles and tendons, which transmitted deep sensibility. These investigators found that following the division of all of the cutaneous nerves to a given area, stimulation with absorbent cotton, the prick of a pin, and warm and cold tubes produced no response, and the two points of the compass could not be discriminated. On the other hand, when this part was touched with any blunt object, the stimulus was at once appreciated, and the point of application could be localized quite accurately. Since all of the nerves which supplied the skin had been divided, the maintenance of this sensibility must have been due to afferent fibers running with the motor nerves. Sherrington² had already demonstrated the existence of such sensory fibers and traced them to the muscles, tendons and joints. The characteristic quality possessed by a part innervated solely by the afferent fibers of a muscular nerve is the appreciation of all stimuli which produced deformation of structure. Pressure or any jarring of the skin is quickly appreciated and, on the whole, well localized. Pressure is not recognized as a sensation when the skin is lifted from the underlying subcutaneous structures, thus demonstrating that this sensation is not due to nerves still remaining in the skin. Pressure produced by means of a Cattell's algometer elicits pain on the affected side, with a lesser degree of force applied than on the sound side. The sensory mechanism in the peripheral nerves was thus found to consist of deep sensibility, capable of answering to pressure and the movement of parts or even producing pain under the influence of excessive pressure; and the superficial sensory system which could be divided into protopathic sensibility, capable of responding to painful cutaneous stimuli and the extremes of heat and cold, and epicritic sensibility, which includes the power of cutaneous localization, of the discrimination of two points and of the finer grades of temperature.

1. Head, Henry: *Studies in Neurology*, London, Oxford Medical Publications **1** and **2**., 1920.

2. Sherrington, C. S.: *On the Anatomical Constitution of Nerves of Skeletal Muscles*, *J. Physiol.* **17**:211, 1894.

In his study of the cranial nerve components, Herrick³ states that while the twelve pairs of cranial nerves are fairly well defined in the human body, they are very diverse in the vertebrate series. Although the facial nerve is predominantly a sensory nerve in the lower vertebrates, it becomes a motor nerve in man with only a vestigial remnant of the sensory component. In studying *Menidia*, Herrick found many instances of distribution of visceral sensory fibers to regions in the mucous membrane of the mouth where there were no taste buds. Chiefly from anatomic evidence, he has classified the facialis as mediating general visceral sensibility. Dixon⁴ has shown rather conclusively in the lower mammals that the facial nerve is a mixed nerve and that its sensory part, the pars intermedia of Wrisberg and the geniculate ganglion, is similar in structure to the sensory portion of other nerves, and that the geniculate ganglion conforms to the cerebrospinal rather than to the sympathetic type.

Primitively, as shown by Johnston⁵ in the lamprey, the facial nerve contains two sensory components; that is, it possesses general cutaneous fibers arising from the geniculate ganglion for the innervation of the skin over the ventrolateral surface of the head below and behind the orbit, and other fibers terminating in the roof and floor of the mouth transmitting other visceral sensation as well as taste. Norris⁶ found a general cutaneous component in *Siren*. As Sheldon⁷ states, the facial nerve of the lower vertebrates, leaving out of consideration the lateral line component of the neuromasts, which is apparently only a part of the facial, is in every case a mixed motor and sensory nerve.

It will be recalled that the facial nerve in man makes its exit from the lateral part of the caudal border of the pons, with the nervus intermedius situated between it and the acoustic nerve. The facial then passes through the internal acoustic meatus and through the canalis facialis in the petrous portion of the temporal bone, emerges at the base of the skull by the stylomastoid foramen, and passes forward through the parotid gland to supply the muscles of the face. In the facial canal the geniculate ganglion is formed at the point where the facial nerve bends backward. From the ganglion, the greater superficial petrosal nerve passes forward through the hiatus canalis facialis

3. Herrick, C. Judson: Doctrine of Nerve Components and Some of Its Applications. *J. Comp. Neurol.* **9**:426, 1899.

4. Dixon, A. F.: Sensory Distribution of the Facial Nerve in Man, *J. Anat. & Physiol.* **33**:471, 1899.

5. Johnston, J. B.: Additional Notes on the Cranial Nerves of Petromyzonts. *J. Comp. Neurol.* **18**:569, 1908.

6. Norris, H. W.: The Cranial Nerves of *Siren Lacertina*, *J. Morphol.* **24**:245, 1913.

7. Sheldon, R. E.: Phylogeny of the Facial Nerve and Chorda Tympani, *Anat. Rec.* **3**:593, 1909.

to the middle fossa of the skull. Behind the tympanum in the lower part of the facial canal, the chorda tympani nerve arises. It enters the tympanic cavity, passes over the membrana tympani and leaves the cavity through the medial end of the petrotympanic fissure to reach the infratemporal fossa. Beneath the external pterygoid muscle it becomes incorporated with the lingual branch of the mandibular division of the trigeminal nerve, and is distributed to the side and dorsum of the tongue in its anterior two thirds.

Prior to the work of Van Gehuchten,⁸ investigators had not been in accord with reference to the origin of sensory fibers contained within the facial nerve. Some had considered them as arising from the trigeminal nerve and reaching the facial through the greater superficial petrosal. Others believed fibers from the auricular branch of the vagus nerve reached the facial. Weigner⁹ described fibers of small caliber, presumably intermedius fibers, in the facial nerve distal to the origin of the chorda tympani.

Streeter¹⁰ showed that at the fifth week the greater superficial petrosal nerve in the human is free from an anastomosis with the trigeminal nerve and that if any fibers grow into it from the fifth nerve they must do so at a very late date. The work of Retzius¹¹ and Lenhossek¹² demonstrated that the geniculate ganglion is composed of nerve cells identical with those of the cerebrospinal ganglions. The central prolongations of these cells enter into the constitution of the intermediate nerve of Wrisberg, while the external prolongations pass into the facial trunk. Van Gehuchten⁸ stained the geniculate ganglion in rabbits in which the facial nerve was sectioned as it left the facial canal. The majority of the cells of the ganglion were found to be normal. However, in every instance, he found a small number of cells which showed evidence of chromatolysis, such as dissolution of chromophilic elements and a considerable displacement of the nucleus. Amabilino¹³ sectioned the facial nerve on one side at the level of the

8. Van Gehuchten, A.: *Recherches sur l'origine réelle des nerfs craniens*, J. de chir. **3**:273, 1898.

9. Weigner: *Ueber den Verlauf des Nervus Intermedius*, Anat. Hefte **29**: 97, 1905.

10. Streeter, G. L.: *The Peripheral Nervous System in the Human Embryo at the End of the First Month*, Am. J. Anat. **8**:285, 1908.

11. Retzius, G.: *Untersuchungen ueber die Nervenzellen der Cerebrospinalen Ganglien und der übrigen peripherischen Kopfganglien*, Arch. f. Anat. u. Physiol. 1880, p. 369.

12. Lenhossek, M. V.: *Das Ganglien geniculi Nervi Facialis und seine Verbindungen*, Beiträge zur Histologie des Nervensystems und der Sinnesorgane, Wiesbaden, 1894.

13. Amabilino: *Sui rapporti del ganglio geniculato con la corda del timpano e col faciale*, Il Pisani **19**:2, 1898.

stylomastoid foramen and on the other side, the chorda tympani during its course in the middle ear. Four fifths of the cells of the ganglion showed definite chromatolysis on the side of the cut chorda tympani. Conversely, on the side of the cut peripheral trunk, about one fifth of the cells showed degenerative changes.

Edgeworth¹⁴ pointed out that the roots of the facial nerve consist of a large fibered portion and of fibers of a smaller diameter. The larger fibers of the nervus intermedius were found to be of all diameters up to 11.2 microns. All of the branches of the facial distal to its exit from the stylomastoid foramen consisted of gray and medullated fibers up to 11.2 microns in diameter. The chorda tympani consisted of medullated fibers of various diameters up to 11.2 microns. From examination of serial sections across the facial trunk in the bone, it appeared to this investigator that of the nerve fibers issuing from the geniculate ganglion, some of the medullated fibers separated off to form the chorda tympani, though a large number of the gray fibers joined the motor part of the facial and became mingled with these fibers. Edgeworth concluded that the muscles supplied by the facial nerve received muscle sensory fibers of all sizes up to a maximum diameter little short of that of their motor fibers.

Rhinehart,¹⁵ working with the albino mouse, stated that the facial nerve corresponds closely to that of other mammals and man. The nervus intermedius portion is composed of afferent fibers having their cell bodies in the geniculate ganglion and efferent fibers which have no connection with the cells of the ganglion. The nervus intermedius of the mouse has three branches; the greater superficial petrosal, the chorda tympani and a facial cutaneous branch. The first two contain both afferent and efferent fibers and the latter, afferent fibers only. These afferent fibers in the mouse terminate in the skin of the external auditory meatus, in the skin of part of the auricle and part of the tympanic membrane. There is no anatomic evidence which justifies the conclusion that such a branch exists in man. Hunt¹⁶ has offered some clinical evidence of the existence of such a nerve.

The experimental evidence offered in the literature points to the existence of afferent fibers within the peripheral facial trunk. The cells of origin of these fibers are in the geniculate ganglion and their internal prolongations pass cranialward in the nervus intermedius. From this evidence, however, the exact function of these fibers has not been

14. Edgeworth, F. H.: On the Medullated Fibers of Some of the Cranial Nerves and the Development of Certain Muscles of the Head, *J. Anat. & Physiol.* **14**:113, 1900.

15. Rhinehart, D. A.: Nervous Facialis of the Albino Mouse, *J. Comp. Neurol.* **30**:81, 1918.

16. Hunt, J. R.: The Sensory System of the Facial Nerve and Its Symptomatology, *J. Nerv. & Ment. Dis.* **36**:321, 1909.

proved. Evidence is at hand which leads to the conclusion that other cranial nerves or their motor divisions carry afferent fibers transmitting impulses from the muscles which they innervate. The origin and termination of the afferent fibers for the extrinsic muscles of the eye are unknown, although we know that such afferent fibers are present in the oculomotor, trochlear and abducens nerves. Johnston¹⁷ has shown that the large unipolar cells of the mesencephalic nucleus of the fifth cranial nerve, which give rise to the fibers of the mesencephalic root of that nerve, are probably sensory in function. Willems¹⁸ and Allen¹⁹ believe that these are sensory fibers to the muscles of mastication.

From the clinical standpoint several investigations have been reported, none of which appears to be sufficiently conclusive to settle the question of the presence of fibers in the fifth or seventh nerves which transmit deep sensibility. During his investigation of taste sensation on the anterior two thirds of the tongue following extirpation of the gasserian ganglion, Cushing²⁰ noted that after some days in association with the return of taste, the movement of a cotton swab over the surface of the tongue gave rise to a peculiar localizable tactile sensation. At the same time, anesthesia to pain, temperature and touch remained absolute. He expressed the view that it was not impossible that the chorda tympani may contain afferent fibers transmitting sensation other than that of taste from the anterior two thirds of the tongue. A patient in whom both the fifth and seventh nerve were paralyzed showed no sensation whatever over the anterior part of the tongue to touch, pain, temperature, taste or the movement of a swab over the surface. In addition, Cushing²¹ carefully studied the boundaries of the sensory loss on the face following trigeminal neurectomy. These observations were made by testing for the loss of superficial cutaneous sensibility only. Whatever afferent fibers may be contained in the facial nerve, he believes reach it by way of the greater superficial petrosal nerve.

Ivy and Johnson²² reported two cases from Frazier's clinic showing a retention of pressure sensibility after destruction of the fifth cranial

17. Johnston, J. B.: The Radix Mesencephalica Trigemini, *J. Comp. Neurol.* **19**:593, 1909.

18. Willems: Les noyaux masticateurs et mésencéphaliques du trijumeau, *Le Névralgiste* **12**:7, 1911.

19. Allen, W. F.: Application of Marchi Method to the Study of the Radix Mesencephalica Trigemini in the Guinea-Pig, *J. Comp. Neurol.* **30**:169, 1919.

20. Cushing, H.: Sensory Distribution of the Fifth Cranial Nerve, *Bull. Johns Hopkins Hosp.* **15**:213, 1904.

21. Cushing, H.: Taste Fibers and Their Independence of the N. Trigemini, *Bull. Johns Hopkins Hosp.* **14**:71, 1903.

22. Ivy and Johnson: Preservation of Deep Sensibility of the Face After Destruction of the Fifth Nerve, *U. of Pennsylvania M. Bull.* **20**:35, 1907.

nerve. One of the cases was a tumor of the gasserian ganglion, and the other was a complete section of the sensory root. No attempt was made to use a standardized or graded type of stimulation. Maloney and Kennedy,²³ in 1911, studied the relation of pressure touch and pressure pain fibers of the face to the facial nerve. For the purpose of their study they arbitrarily divided the facial nerve into four parts; (1) the peripheral portion of the nerve after its exit from the stylomastoid foramen; (2) that part of the nerve between the Fallopian aqueduct and the stylomastoid foramen; (3) that portion lying within the aqueduct, and (4) the central portion from the facial nucleus to the canal. Of their clinical material, there were eight cases of section of the second and third divisions, one case of section of the second division of the trigeminal nerve and ten cases of complete seventh nerve paralysis. For the purpose of their tests, they used an esthesiometer of 2 gm. weight and increased the pressure exerted by the addition of weights to 65 gm. They found uniformly that loss of sensation in the conjunctiva included all types of stimulation. Five cases showed that complete section of the facial nerve distal to the stylomastoid foramen may occur without loss of pressure pain sense. However, these were tested in the presence of cutaneous sensibility. These authors stated that this same finding may occur with a section of the nerve as high as the entrance of the facial nerve into the middle ear. On the other hand, pressure pain sensation was lost with a lesion in the Fallopian canal. They concluded that pressure pain sense is affected after intracranial interference with the trigeminal nerve, a procedure which they believe probably damages the functional activity of the sensory fibers of the facial nerve. This loss was permanent in some instances, so they reasoned that the fibers which join the facial nerve in the Fallopian aqueduct must first enter the region of the trigeminal roots. On the other hand, the constancy, severity and permanency of loss of all sensation in the eye indicated that the pressure pain fibers were invariably concentrated in the trigeminal nerve. It is obvious that such an explanation necessitates a fanciful anatomic pathway for fibers transmitting deep sensibility.

Applying Head's views on the peripheral nervous system to the facial nerve, Mills²⁴ argued that the afferent system concerned with deep sensibility, including pressure and postural senses, may be conveyed cranialward in the facial trunk, leaving it somewhere before it enters the cerebrospinal axis. He believed that the geniculate ganglion and its distal and proximal roots could be regarded on the basis of

23. Maloney, W. J., and Kennedy, R. F.: The Sense of Pressure in the Face, Eye and Tongue, *Brain* **34**:1, 1911.

24. Mills, C. K.: Sensory Functions Attributed to the Seventh Nerve, *J. Nerv. & Ment. Dis.* **37**:273, 1910.

homologues between spinal and cranial ganglions, such as the sensory portion of the facial nerve. He further predicated that if deep sensibility is relegated to a separate afferent system, the fibers should run with the motor fibers, although in a different direction. This would bring the facial and the motor portion of the trigeminal nerve into harmony with what Head believes to be the case in the spinal system. In Mill's work he deemed it essential that the condition as to cutaneous sensibility of the epicritic and protopathic types should be carefully determined. He concluded from his work that the sense of pressure pain was represented in the trigeminal and not in the facial nerve. In all cases of facial nerve paralysis investigated by this author, no alteration in the sense of pressure pain could be determined. Mills went a step further and argued that if Head's hypothesis held true with reference to pressure pain sense, then postural and passive movement sensation should be subject to the same alterations. He tested facial movements with a faradic current when cutaneous areas were totally anesthetic. Under these conditions his patients did not appreciate movement in the facial muscles. His final conclusion was that if the afferent fibers concerned with the sense of pressure posture and passive movement pass with the facial nerve, they diverge immediately after entering the cranium and join the sensory branches of the trigeminal nerve before the latter reach the gasserian ganglion.

Dana²⁵ believed that every form of sensibility is lost when the trigeminal nerve is cut close to the ganglion. This author reported a case in which the mandibular division of the fifth nerve was cut. The patient had lost sensibility to touch, pain, temperature, deep pressure sense and lacked recognition of movements of the facial muscles.

Davies²⁶ stated that deep sensation is invariably absent in regions in which there is a total loss of skin sensibility; in other words, he believed that sensory fibers from the muscles of the face travel in the trigeminal nerve. If the soft tissues were pressed upon the underlying bones, the patients said that they could feel a slight touch; yet if asked to indicate the spot, localization was imperfect. Postural changes of muscles produced by faradization or gentle traction on the lip or nose were not noticed, nor were the patients aware when the cheek became distended.

Kidd²⁷ asserts that if the seventh cranial nerve contains pressure pain fibers, they pass centrally by way of the pars intermedia. The

25. Dana, C. L.: Question of Protopathic and Epicritic Sensibility and the Distribution of the Trigeminal Nerve, *J. Nerv. & Ment. Dis.* **33**:577, 1906.

26. Davies, H. Morriston: Functions of the Trigeminal Nerve, *Brain* **30**:219, 1907.

27. Kidd, L. J.: Alleged Sensory Cutaneous Zone of the Facial Nerve of Man, *Rev. Neurol. & Psychiat.* **12**:393, 1914.

small size of this afferent root and its ganglion might suggest that this was impossible, for it has to transmit taste fibers as well. On the other hand, the deep afferent system needs a far smaller number of fibers for the transmission of its impulses than does the cutaneous epicritic system. Therefore, the afferent trigeminal tract may be looked on as an epicritic and protopathic nerve par excellence, whereas the afferent facial nerve probably conveys the relatively low grade deep sensibility fibers.

In reporting a case of tumor of the gasserian ganglion, Spiller²⁸ stated that tactile sensation was completely lost in the entire distribution of the fifth nerve, provided no pressure was used. The cornea was absolutely insensitive and pin prick sense was lost. He adds that in some cases of removal of the gasserian ganglion all sensation, including pressure sense, is lost, at least for a period following operation, but in two cases he had noted the preservation of pressure sense. Spiller believes that complete loss of all forms of sensation after excision of the gasserian ganglion does not make the transmission of certain forms of sensation through the seventh nerve impossible. On the other hand, deep sensation of the face may be interfered with by an operation on the fifth nerve.

METHODS

The methods for investigating the question of deep sensibility of the face may be divided into the examination of clinical cases and experiments performed on animals. The clinical cases consisted of trigeminal neurectomies and cases in which there was a simultaneous loss of the functions of both the fifth and seventh cranial nerves. Superficial sensation was tested in each instance by the use of an algesimeter of 15 gm. weight, a wisp of absorbent cotton and test tubes containing hot and cold water. Deep sensibility was investigated by the use of a Cattell algometer, the scale of which was graduated up to 15 kilograms. A reading was taken when the patient voluntarily manifested pain on pressure. Several readings were taken in each case over the ophthalmic, maxillary and mandibular areas of both the affected and unaffected sides.

Cats were used throughout the work as the experimental animals. Intracranial division of the sensory root of the trigeminal nerve or complete destruction and avulsion of the gasserian ganglion was performed on twenty-five animals. Aseptic measures were followed out in all operative procedures and the animals allowed to live varying lengths of time following the operation. Stimulation by means of an electric current was used to test the superficial and deep sensibility in the animals. Following the study of the sensation in each animal, they

28. Spiller, W. G.: Tumor of the Gasserian Ganglion. *Am. J. M. Sc.* **136**: 712, 1908.

were again operated on, and the facial nerve on the same side was sectioned at its exit from the stylomastoid foramen. The central end of the facial nerve was then stimulated with a sterile electrode in five of the animals. Again the animals were allowed to recover, and superficial and deep sensation of the face was tested. An electrode with needle points was used so that in testing for deep sensibility the points could be thrust beneath the cutaneous tissue into the muscles and on to the bones of the face. Postmortem examinations were made on each animal to determine the extent of the lesion involving the fifth nerve ganglion and definitely to prove that the seventh nerve was completely cut. In another series of eight animals, the facial nerve on the left side was cut at the stylomastoid foramen, and the lingual nerve containing the chorda tympani fibers was sectioned high on the right side. These animals were allowed to live about one week, when they were sacrificed and both geniculate ganglions were removed. A normal ganglion was carried through the staining preparations in each case in addition to the pathologic specimens, so that changes which might be produced by staining technic could be controlled. Toluidin blue-erythrosin was used as the staining fluid to determine ganglion cell degeneration changes, if such existed.

EXPERIMENTAL ANIMALS

Of twenty-five experimental animals, twenty-four showed responses from stimulation with an electric current which were exactly similar. It will suffice therefore to give in detail the protocol of one of these. The explanation of the responses obtained on the exception will be clearly seen in the protocol to be given.

The same technic used in producing the lesions of the fifth and seventh cranial nerves was adhered to in each animal. Under ether anesthesia the hair was removed from the side of the head to be operated on. The field of operation was painted with iodine and blocked off by sterile towels and sheets. A linear incision was made from the zygoma upward just in front of the ear. The fascia of the temporal muscle was incised and the fibers split in the direction of their course. They were held apart by a mastoid retractor, exposing the underlying bone. After all bleeding was controlled and with the zygomatic process as a base, a trephine opening was made through the bone. By the aid of small brain sponges, the dura was carefully separated from the floor of the middle fossa. An opening into the dura mater was carefully avoided. The temporal lobe was retracted upward and gauze dissection continued across the floor of the middle fossa to the gasserian ganglion. Considerable oozing was commonly encountered as the ganglion was exposed, but this could be controlled by pressure. After the ganglion had been completely exposed, a blunt nerve hook was passed beneath

it near the entrance of its sensory root. With a sharp right angled knife the sensory root was then divided. If trouble was experienced in visualizing the sensory root, the ganglion was completely avulsed. After hemostasis was secured, the wound was closed in layers and a collodion dressing applied. The animals recovered promptly and were tested for the presence of superficial sensation by a strong faradic current. These examinations were frequently repeated and the responses noted. After ten days to two weeks, a second operation was performed at which time the seventh nerve on the same side was cut at its exit from the stylomastoid foramen. After the operative recovery was complete, sensation was again tested by the electric current. Needle electrodes applied to the skin of the face with the secondary coil of the inductorium at zero was the unit of stimulation used to determine the presence or absence of superficial sensation. These electrodes could then be thrust through the skin into the facial muscles and stimulation effected to determine the presence or absence of deep sensibility.

Each animal was subjected to a postmortem examination to determine the extent of the lesion of both nerves. The geniculate ganglions of Animals 9, 11, 26, 27, 28, 29, 30 and 31 were studied for cell changes. In Animals 26 to 31 inclusive, the right lingual and the left facial nerves were cut and the respective geniculate ganglions studied by the toluidin blue-erythrosin method.

Protocol of Animal 9.—On Feb. 23, 1922, under ether anesthesia, the left sensory root of the trigeminal nerve of a female cat, weighing 3 kilograms, was severed. Hemorrhage was slight and a good exposure was obtained. The operative recovery was very prompt. On February 27, superficial stimulation with a unit of faradic current produced no response over the left half of the face, mucous membrane of the mouth or of the cornea. On thrusting the electrodes beneath the skin and into the muscles of the face, stimulation with the same strength of current produced a wild cry, struggling, pulling away and snapping. These responses were corroborated on March 1, 2, 3 and 5. On March 6, the left facial nerve was cut at the stylomastoid foramen. The nerve was cut some distance from the foramen and the central end carefully and completely isolated. It was then stimulated with a faradic current using a sterile electrode. A weak current caused the cat to cry out, and the right pupil dilated. A stronger stimulus produced struggling and an attempt to bite the investigator. The right pupil became constricted, but no change was noted in the left eye. The nerve was then cut close to the foramen. The animal again made a complete operative recovery. On March 9, at which time the facial paralysis was quite marked, the animal was again stimulated with an electric current. No response was obtained from superficial or deep stimulation on the left half of the face. These responses were corroborated on March 13 and 20 and on April 6. On April 7, the animal was killed and postmortem examination revealed a complete division of the fifth nerve posterior to the gasserian ganglion, as well as a complete severance of the facial nerve at the stylomastoid foramen.

Protocol of Animal 20.—The left gasserian ganglion and sensory root of a male cat, weighing 3.5 kilograms, were exposed, June 13, 1922. There was profuse bleeding from the cavernous sinus when an attempt was made to expose the sensory root more thoroughly. This was so troublesome that rather than prolong the operation, a nerve hook was placed beneath the ganglion and avulsion and destruction attempted. The animal made a very slow operative recovery, showing a definite left cerebellar ataxia for five days. On June 21, the animal was tested with a faradic current. The cornea was sensitive to the stimulation and to touch with a wisp of cotton. There was a small area the size of a ten cent piece over the maxillary eminence in which superficial stimulation produced no response. Elsewhere superficial sensation was present, including the mucous membranes of the nose and mouth. Stimulation with the electrodes placed beneath the skin and into the muscles of the face everywhere produced struggling and crying out. On June 26, the facial nerve was cut at the stylomastoid foramen. On June 29, 30 and July 2, several observations were made on this animal. Superficial stimulation with the faradic current produced the same response over identical areas as before. Stimulation deep beneath the skin produced struggling and crying out over the entire left half of the face. Postmortem examination showed that the lesion produced in the gasserian ganglion was quite incomplete. The sensory root was intact. There was a small lesion in the center of the ganglion which could be seen grossly. It was quite evident that division had not been accomplished.

CLINICAL CASES

The cases reported below for the most part are patients from the service of Dr. Allen B. Kanavel at Wesley Memorial Hospital. Each of the patients was completely examined, including a thorough neurologic examination. The superficial and deep sensation of the face was particularly investigated prior to operation, and each case was established as one of major trigeminal neuralgia. Following operation, each patient was again examined several times over periods varying from two weeks to three years after operation. Figure 1 is a composite diagram of the superficial sensory loss found in these cases. In any given case the line of sensory loss varied only about 0.5 cm. internal or external to this line.

CASE 1 (Mrs. C. S.).—On Jan. 15, 1922, the sensory root of the left trigeminal nerve was divided for the relief of a persistent trigeminal neuralgia which had been present for twelve years. Six alcoholic injections had been given previously, all of which had given relief. The last injection produced anesthesia and relief of pain for only one month. The dolorigenetic zone in this patient was at the left angle of the mouth, where stroking the skin with a wisp of cotton or a blast of cold air would produce the pain. Chewing and talking also excited pain from which the patient could get no relief. Following the operation sensation to pin prick, touch and temperature stimuli was lost over the entire half of the face bordered by a line corresponding to the average. The algometer readings over the left half of the face were: ophthalmic, 5 kgm.; maxillary, 2 kgm.; mandibular, 2 kgm. The corresponding readings over the right half were 2, 2 and 2 kilograms, respectively. The patient voluntarily described the sensation produced by the algometer as a crushing pain, and that

it seemed confined to the point of pressure on the left side, but on the unaffected side the pain was more radiating in character. These findings were corroborated on April 9, 1922.

CASE 2 (Mrs. F. C.).—In this case, division of the sensory root of the left trigeminal nerve had been performed six months previous to examination for the purpose of this work. When the operation was performed it had been quite possible to preserve the motor root. The boundary of the loss of superficial sensation was within the normal limits. However, it may be added that the cervical nerves did not overlap into the mandibular areas as far as in some of the other cases. The patient had perfect function of the left masseter and pterygoid muscles. On examination with the algometer, pressure pain responses

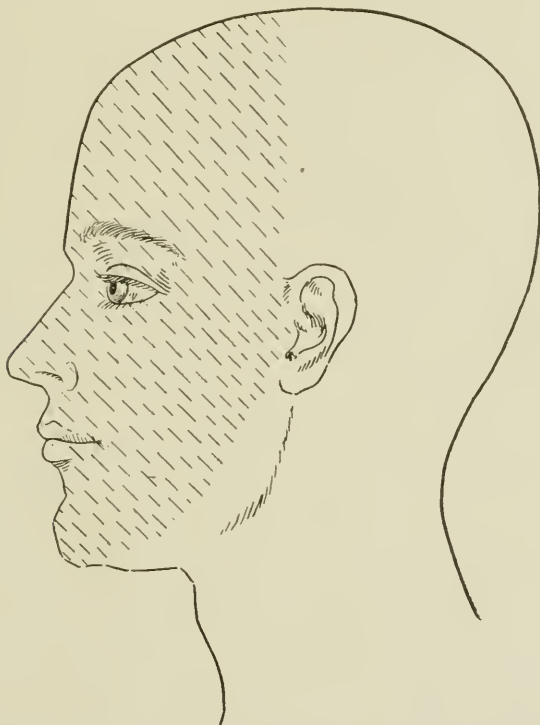


Fig. 1.—A complete diagram showing the loss of superficial sensation in ten cases of division of the sensory root of the trigeminal nerve for trigeminal neuralgia.

were present over both sides of the face. The readings were: left ophthalmic, 4 kgm.; maxillary, 2 kgm., and mandibular, 3 kgm.; right ophthalmic, 3 kgm.; maxillary, 2 kgm., and mandibular, 2 kgm. This patient described the sensation on the affected side as that similar to pressure on a sore tooth. No difference was noted in the radiation of the pain on the two sides. The two point discrimination test produced a correct response from the right side when the points were separated 2.5 cm. and when they were applied transversely. Correct responses could not be obtained on the left side with the points separated 4 cm.

CASE 3 (Mrs. L. M.).—A true major trigeminal neuralgia had been present on the right side for three years. Several deep alcohol injections had been attempted without the production of sensory loss, and consequently without relief of pain. The dolorigenetic zone in this patient was situated on the mucous membrane of the right cheek near the angle of the mouth. The pain would spread to involve the maxillary and ophthalmic division areas. The patient was examined one year following her operation, during which time she had had no recurrence of her pain. The right side of the face showed absolute loss to pin prick, temperature and touch sensation. The algometer readings were: right ophthalmic, 5 kgm.; maxillary, 3 kgm., and mandibular, 2 kgm.; left ophthalmic, 5 kgm.; maxillary, 3 kgm., and mandibular, 3 kgm. The patient described the sensation as an aching pain and could not distinguish any difference in severity on the two sides.

CASE 4 (Mrs. S. W.).—This patient was operated on two years ago (1920) for the relief of a right-sided trigeminal neuralgia. The records show that the patient had a facial paralysis of the peripheral type on the right side immediately following operation. This is an example of those fortunately uncommon paralyses which are most satisfactorily explained on the basis of traction on the greater superficial petrosal nerve at the time of operation and in which the patient more or less promptly recovers. At the time of my examination, no evidence of facial nerve involvement was present. The loss of superficial sensation was bounded laterally by a line situated well in front of the ear. The overlap from the cervical nerves was more extensive than the average. Pressure pain sensibility was present on both sides, as shown by these algometer readings: right ophthalmic, 2 kgm.; maxillary, 3 kgm., and mandibular, 2 kgm.; left ophthalmic, 2 kgm.; maxillary, 2 kgm., and mandibular, 2 kgm. The sensation was described as a "heavy pressing." Two point discrimination sense could not be accurately determined on the right side.

CASE 5 (Mrs. M. F.).—This patient had suffered with a left major trigeminal neuralgia for eight years. She had developed a habit of sucking in the left cheek between her gums to afford some slight relief. Talking, particularly, was an exciting factor. The terrific pain which had never been relieved by alcoholic injections had brought about a marked neurotic element. The left sensory root was sectioned, and immediately following the operation for four days there was a flaccidity of the left facial muscles. This had entirely disappeared at the time of her discharge two weeks later. The algometer readings made on the fourth day postoperatively were: right ophthalmic, 2 kgm.; maxillary, 5 kgm., and mandibular, 3 kgm.; left ophthalmic, 10 kgm.; maxillary, 12 kgm., and mandibular, 8 kgm. At the time of her discharge the readings were the same on the right side while on the left they were: ophthalmic, 5 kgm.; maxillary, 4 kgm., and mandibular, 3 kgm. These readings are somewhat higher than in the cases previously cited in comparison with the unaffected side. The facial nerve was no doubt affected in this case, but the temporary disturbance rapidly cleared up, and with it the algometer readings became more nearly normal. The patient signified that pressure had been applied at a lower reading, but the element of pain did not enter until the algometer had been depressed to the readings given. A round smooth piece of wood was introduced into each nares of this patient and pressure exerted laterally. The lacrimal reflex was obtained on the right side, as well as the sneezing reflex. On the left, the sneezing reflex was absent, but a lacrimal reflex was present.

CASE 6 (Mrs. I. M.).—The dolorigenetic zones in this patient's face were in the upper cheek on the right side, particularly on the mucous membrane opposite the second molar tooth. Difficulty was experienced in exposing the

sensory root because of adhesions previously induced by alcoholic injections. The ganglion was therefore completely avulsed. Sensation to pin prick, touch and temperature sense was lost over an area corresponding to the average loss. The algometer readings were: left ophthalmic, 2 kgm.; maxillary, 2 kgm., and mandibular, 2 kgm.; right ophthalmic, 3.5 kgm.; maxillary, 2 kgm., and mandibular, 3 kgm. The sensation produced on the affected side was a distinct one of a heavy, severe pressure, which did not spread. The patient could not discriminate one or two points over the affected side. Pressure inside the right nostril was felt, but not as distinctly as on the left. This pressure produced a lacrimal reflex on both sides.

CASE 7 (Mrs. M. S.).—This patient was operated on in July, 1922, for the relief of a major neuralgia, which had been present on the right side for two years. No alcohol injections had been given at any time. Following operation the patient had a paralysis of the right frontalis muscle, which was due to involvement of the frontal branch of the facial nerve in the operative incision. There was complete loss of superficial sensation in the trigeminal area. The algometer readings were: right ophthalmic, 14 kgm.; maxillary, 5 kgm., and mandibular, 3 kgm. The corresponding readings on the left side were: ophthalmic, 4 kgm.; maxillary, 3 kgm., and mandibular, 3 kgm. It will be seen that the reading over the right side of the forehead is high. Heavy pressure could be felt at all times, but it was not particularly painful. The readings were normal over the rest of the face.

CASE 8. (Mrs. H. C.).—A right trigeminal neuralgia had been present for six years. The first deep alcohol injection gave relief for two years. Later injections were unsuccessful. Following division of the sensory root, complete loss of superficial sensation was present over the usual area, except that there was a fairly wide overlap from the cervical nerves. The algometer readings were: right ophthalmic, 4 kgm.; maxillary, 5 kgm.; mandibular, 3 kgm.; left ophthalmic, 3 kgm.; maxillary, 2 kgm., and mandibular, 2 kgm. The sensation was described everywhere as a heavy crushing feeling, but was not distressingly painful.

CASE 9 (Mrs. T. H.).—This patient had suffered almost continuously since February, 1921 with a severe neuralgia of the left side of the face. Eating, talking and touching the face over the mandibular area caused an exacerbation of pain which spread over the entire fifth nerve distribution. A palpable thyroid gland was present, but there were no symptoms of pressure or of toxemia. The blood pressure was: systolic, 236; diastolic, 120. The patient withstood the operation very well, and there was an immediate relief from pain. The loss of superficial sensation here corresponded to the usual area. The algometer readings were: right ophthalmic, 2 kgm.; maxillary, 1 kgm., and mandibular, 1 kgm.; while on the left side they were ophthalmic, 2 kgm.; maxillary, 3 kgm., and mandibular, 2 kgm. The patient winced, but said that she did not know how to describe the sensation of the pain produced.

CASE 10 (Mrs. C. H.).—A right-sided major neuralgia which had been present for twelve years had brought this patient to the point of extreme emaciation and exhaustion. She had had four deep alcohol injections, but only one had been successful. Relief from pain lasted four months. On operation, dense adhesions were present between the dura mater and the floor of the skull. The ganglion was exposed with difficulty and eventually was avulsed. The sensory loss to pin prick, touch and temperature stimuli was within the usual borders. The patient could not accurately discriminate between one and two points applied to the face. The algometer produced a pain which was terrifying

to the patient. On the right side the readings were: ophthalmic, 3 kgm.; maxillary, 4 kgm., and mandibular, 2 kgm.; while on the left side the corresponding readings were: 3, 3 and 3 kgm. This patient noted that on the right side the pain did not radiate.

CASE 11 (Mrs. C. B.).—Four years before this patient developed a left peripheral paralysis of the facial nerve, which rapidly became better within two weeks. Subsequently, she began to have a neuralgic pain in the left side of her face, which began over the maxillary process and spread over the distribution of the maxillary and ophthalmic divisions of the trigeminal nerve. Cold air, cool water and stroking the face produced exacerbations of the pain. She suffered severely for a year and was referred by a neurologist for a neu-



Fig. 2 (Case 13).—Facial paralysis in the left frontal area. There was a complete loss of pin prick, touch and temperature sensation in the ophthalmic and maxillary division areas. The numbers within the circles represent the average readings of the algometer before pressure pain was produced.

rectomy as having a case of major neuralgia. Three years have elapsed since her operation. There is a complete loss of superficial sensation over the distribution of the left trigeminal nerve. Pressure with the algometer, however, produced definite pain localized to the point of stimulation over the left side of the face. The algometer readings were: right ophthalmic, 4 kgm.; maxillary, 3 kgm., and mandibular, 3 kgm.; left ophthalmic, 3 kgm.; maxillary, 2 kgm., and mandibular, 2 kgm. At this time, there was no evidence of any lesion of the left facial nerve. The motor division of the left fifth cranial nerve is paralyzed. The nasal mucous membrane on the left side is insensitive to pin prick, touch and temperature stimuli. Pressure with a blunt object within the nose produced a definite lacrimation in the left eye.

CASE 12 (Mr. C. R.).—This patient was examined in the neurological ward of the Cook County Hospital where he had entered complaining of bilateral disturbance of sensation of the face, diplopia and loss of taste sensation. The sensory loss in his face had its onset about a year previously and had begun in the region of the gums of the lower front teeth. The sensory loss began with a disturbance of heat and cold sensation, but as its area increased in size, the loss to pain became pronounced. At the time of examination, complete analgesia was present to pin prick stimulation over both trigeminal areas. Temperature sense was lost on the right side and was considerably impaired on the left. Touch sense, tested with a wisp of absorbent cotton was everywhere present. The patient had difficulty in distinguishing between sweet and sour stimuli over the anterior two thirds of the protruded tongue. No neuralgiform pain was complained of and the patient had no facial spasms. Argyll Robertson pupils were present, and the right pupil was larger than the left. The motor facial nerve was not clinically involved. Station and gait were normal and active, and passive movements were good. No paresis nor paralysis was present in any of the extremities. Pressure pain sense was present over both sides of the face as determined by pressure with the algometer. The readings were: right ophthalmic, 6 kgm.; maxillary, 5 kgm., and mandibular, 5 kgm.; left ophthalmic, 5 kgm.; maxillary, 5 kgm., and mandibular, 5 kgm.

CASE 13 (Miss C. L.).—This young Irish girl had been operated on two times at another hospital for the relief of a left-sided neuralgia. The scars of two osteoplastic flaps were visible. Although the patient had a total anesthesia to superficial cutaneous stimuli in the area of the ophthalmic and maxillary divisions, she still complained of severe pain in the mandibular area. This pain had its origin about the gums of the lower teeth on the left side. The left half of the forehead had been paralyzed since the branch of the facial nerve which supplies the frontal muscle had been cut in the skin incisions. Fig. 2 shows this paralysis quite clearly. The lower facial muscles were unaffected. The algometer readings were: right ophthalmic, 2 kgm.; maxillary, 2 kgm., and mandibular, 1 kgm.; left ophthalmic, 15 kgm.; maxillary, 2 kgm., and mandibular, 1 kgm. Pressure of 15 kgm. over the forehead, where both the fifth and seventh cranial nerve supply was absent, produced no sensation. Elsewhere over the face the algometer produced definite painful responses.

CASE 14 (Mr. F. H.).—This patient had been under treatment in the Ear Dispensary of the Northwestern University Medical School for many months for tinnitus, vertigo and loss of hearing on the right side. He rather suddenly developed a peripheral paralysis of the right facial nerve. Following this, over a period of eighteen months, the right trigeminal and glossopharyngeal nerves had also become involved (Fig. 3). The marked facial paralysis and the trophic lesion of the right eye are seen distinctly. On electric stimulation there is no response of the right facial muscles to either the faradic or galvanic currents. Sensation on the affected side is entirely lost to pin prick, touch and temperature sense. Pressure over the right half of the face with the algometer produces no sensation, even though the maximum stimulation of 15 kgm. is used. On the left side, pain is produced with a pressure of: ophthalmic, 3 kgm.; maxillary, 2 kgm., and mandibular, 3 kgm. Pressure with the algometer on the outstretched tongue produces pain, although sensation to the usual superficial stimuli, as well as taste sensation, is entirely lost over the right half of the tongue.

Figure 4 is a diagram on which have been placed the average readings obtained with the algometer in ten of the foregoing cases. Reading from left to right, the corresponding numbers on the left and right halves of the face represent a given case.

COMMENTS

From the evidence which has been gathered in the literature it appears to be well established that in the lower vertebrate series the seventh cranial nerve contains a sensory component, the fibers of which



Fig. 3 (Case 14).—Trigeminal and facial nerve lesions on the right side. The numbers within the circles represent the average readings of the algometer before pressure pain was produced.

should probably be classed as general visceral afferent, since the muscles supplied by the facial nerve are of branchial origin. As one ascends in the mammalian series to man, the sensory portion of this nerve decreases in size. In man it is represented in the *pars intermedius* or the nerve of Wrisberg. It is here comparatively only a vestigial remnant. It can be easily understood, therefore, why the facial nerve should have been considered practically a pure motor nerve for so long a period. The demonstration that the taste fibers from the anterior two thirds of the

tongue course cranialward in the chorda tympani branch, the cells of which are in the geniculate ganglion, were signal observations. The most logical pathway for these afferent fibers cephalad from the geniculate ganglion would be within the nervus intermedius. These facts have been corroborated often and may be accepted. There need be no fanciful explanation which carries these impulses through the greater superficial petrosal to the gasserian ganglion, when a more direct anatomic course can be proved.

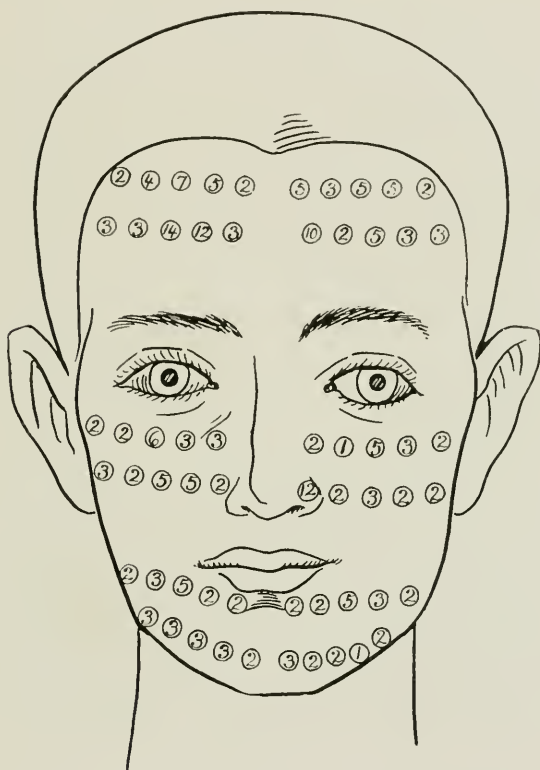


Fig. 4.—Average readings of the algometer producing pressure pain sensation in ten cases are shown within the circles. Reading from left to right in each area, the numbers represent the readings on the right and left sides in a given case.

In spite of these observations and those of Retzius, Lenhossek, Amabilino and Van Gehuchten on the histology of the geniculate ganglion cells and their changes after experimental lesions, the presence in the facial nerve of sensory fibers from the facial muscles has never been established. A review of the literature shows the opinion of the majority to be that destruction of the trigeminal nerve supply to the face produces a loss of all types of sensory stimulation. In most

instances, deep sensibility has been tested in an unstandardized manner, and results obtained from deep stimulation in the presence of perfect superficial cutaneous sensation are of little value. The supposed presence of muscle sensory fibers within the fifth cranial nerve has consequently called forth many explanations which have been quite unsubstantiated from an anatomic standpoint. These have predicated the supply of the facial muscles with sensation directly from the trigeminal nerve, or the presence of these fibers within the facial nerve

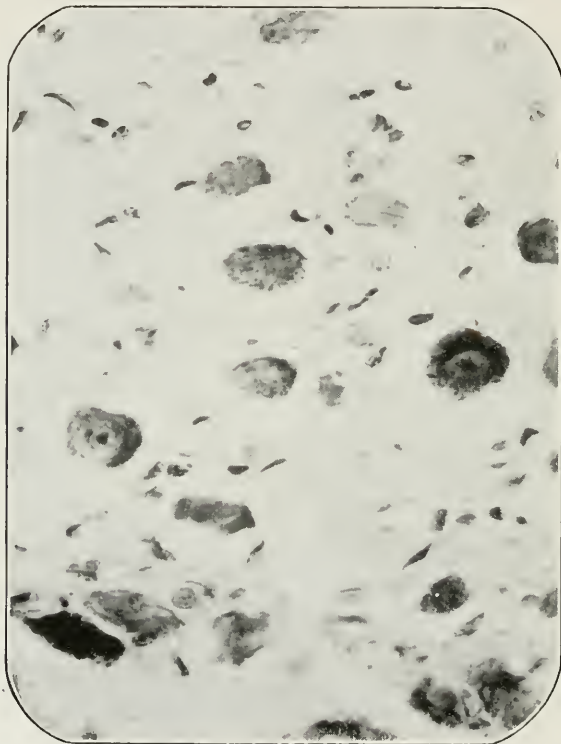


Fig. 5.—Section of geniculate ganglion of a cat in which the facial nerve had been severed at the stylomastoid foramen. Toluidin blue-erythrosin stain.

peripherally and later reaching the fifth through the greater superficial petrosal or by some other unexplained pathway within the cranial cavity. On the contrary, the clinical observations of Kidd and Ivy and Johnson support the view that the facial nerve carries muscle sensory fibers and that the trigeminal is an epicritic and protopathic nerve par excellence. There has not been offered any experimental evidence to corroborate any of these clinical observations.

The foregoing animal experiments must lead one to the recognition of certain facts. When the trigeminal sensory root is completely cut

or the ganglion thoroughly avulsed, there is a complete loss of response on the corresponding half of the face, cornea and mucous membrane of the nose and mouth to superficial stimulation with a strong and painful faradic current. However, if the sharp pointed electrodes are thrust beneath the skin into the facial muscles or on to the bone and stimulation effected, there is an unquestioned response. Further, if the facial nerve to the same side is cut, producing a definite peripheral facial paralysis, neither superficial nor deep faradization produces a response.

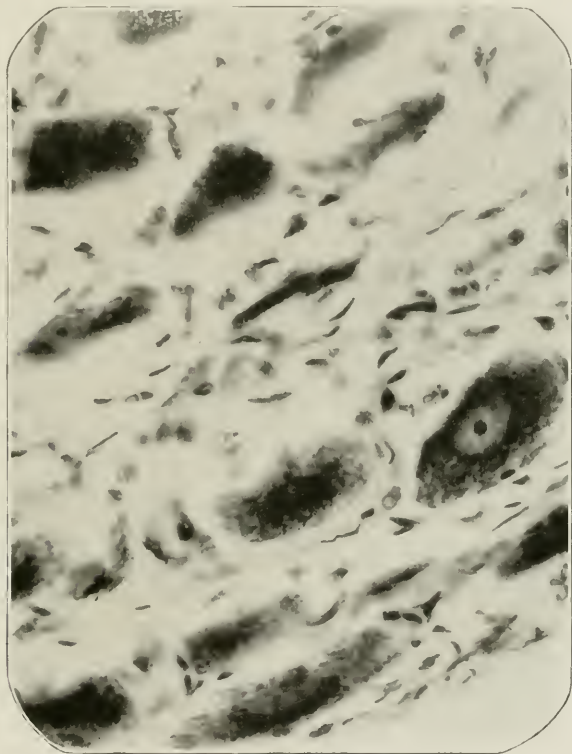


Fig. 6.—Section of geniculate ganglion of a cat in which the chorda tympani nerve had been destroyed. Toluidin blue-erythrosin stain.

This certainly suggests the presence of afferent fibers within the peripheral facial nerve supplying the muscles of the face. Further proof of this may be obtained by examining the geniculate ganglions of animals in which the facial nerve has been cut at the stylomastoid foramen. Figure 5 is a toluidin blue-erythrosin stain of such a ganglion, while Figure 6 is a like stain of the geniculate ganglion of an animal in which the chorda tympani nerve had been destroyed. The ganglion of the cut peripheral facial nerve shows a smaller number of cells with chromatolysis than does the ganglion of the cut chorda tympani. Never-

theless, in the former case chromatolytic cell changes cannot be denied. The work of Van Gehuchten and Amabilino supports these findings.

It is quite true that statements as to the presence or absence of sensation following experimental lesions in animals cannot be conclusive. On the other hand, such facts in conjunction with clinical evidence bear weight. Each of the patients on whom a trigeminal neurectomy had been performed showed a loss of superficial sensation corresponding to the accepted area of the trigeminal supply. In addition, each of the patients showed the definite presence of pressure pain sensation, and the algometer readings on the affected side were almost identical with those on the normal half of the face. When differences were present, there was always a higher reading on the side of the lesion. Radiation of the pain is absent on the side of the cut nerve, as is one and two point discrimination sensibility. Cases tested for the lacrimal reflex by pressure within the nares showed that it was present. In addition, we were quite fortunate in being able to observe one case in which there was a concomitant total loss of the fifth and seventh cranial nerves on one side and another case in which this condition was present over the ophthalmic division of the fifth nerve. In the first instance, pressure pain could not be produced with 15 kg. of pressure over any point on the right half of the face, the side of the lesion. In the second case, the same observation was made concerning the region over the forehead where the fifth and seventh nerve supply was lost.

Here, then, exists experimental and clinical evidence that muscle sensory fibers to the facial muscles are contained within the peripheral trunk of the facial nerve, the cells of which exist within the geniculate ganglion. Applying Head's theory of the sensory mechanism in the peripheral nerves to the sensory supply of the face, it would mean that the trigeminal nerve carries the afferent epicritic and protopathic impulses, while the facial nerve transmits the impulses of deep sensibility from the face.

CONCLUSIONS

1. Pressure pain sensibility in the face is retained following trigeminal neurectomy or ganglionectomy, although all superficial cutaneous sensibility is lost.
2. Afferent fibers which transmit pressure pain sensation from the face exist within the facial nerve trunk.
3. The cells of origin of these afferent fibers exist within the geniculate ganglion, and their internal prolongations pass cephalad within the *nervus intermedius*.
4. The facial nerve is, therefore, a mixed nerve in man, and contains a general visceral sensory component.

DISCUSSION

DR. LEWIS J. POLLOCK: I should like to ask Dr. Davis one or two questions, but before doing that I would like to call Dr. Patrick's attention to something I think we have observed together. In peripheral facial palsies, the patients were unable to feel muscle movement produced by galvanic stimulation, unless it was very painful.

I would like to ask Dr. Davis whether he had occasion to examine the pressure sense in the tongue after a section of the sensory root of the fifth, or gasserectomy. It would appear to me that if the fibers for deep sensibility are carried along with a motor nerve to the muscle which it innervates, pressure sense should be supplied to the tongue by the hypoglossal; and therefore I should expect that the deep pressure sense would not be lost in the tongue following a gasserectomy.

DR. LOYAL E. DAVIS: I did not examine the patients for the presence of deep sensibility in the tongue, but to suppose that the chorda tympani fibers carry this sensation seems to be rather far fetched. It would seem more logical to place the fibers transmitting deep sensation from the tongue in the hypoglossal nerve.

I did not report six or seven cases of early peripheral facial paralysis which I examined for the determination of muscle movements, because the patients to my mind were not intelligent enough to be able to distinguish the difference between the sensation produced by the stimulation with the electric current and the movements of the facial muscles. I feel, however, that sensation produced by muscle movements is lost with pressure pain sensibility with a lesion of the facial nerve.

AFFERENT IMPULSES OF THE TRIGEMINAL NERVE

THE INTRAMEDULLARY COURSE OF THE PAINFUL, THERMAL AND TACTILE IMPULSES *

MARGARET WILSON GERARD

CHICAGO

CONTENTS

- I. INTRODUCTION.
 - 1. The Anatomy of the Trigeminal Nerve.
 - 2. Functional Divisions of the Spinal Trigeminal Tract.
 - 3. Blood Supply for the Spinal Trigeminal Tract and Nucleus.
- II. CLINICAL DATA.
 - 1. Symptom Complex of Occlusion of the Posterior Inferior Cerebellar Artery.
 - 2. Clinical Cases of Its Occlusion.
 - 3. Cases with Other Lesions.
 - 4. Summary and Discussion of Cases.
- III. EXPERIMENTAL WORK.
 - 1. Introduction.
 - 2. Surgical Technic.
 - 3. Methods of Experimentation.
 - 4. Experimental Results.
 - 5. Comment.
- IV. CONCLUSIONS.

I. INTRODUCTION

ANATOMY OF THE TRIGEMINAL NERVE

The trigeminal nerve is composed of a large sensory division whose unipolar cells are in the Gasserian ganglion, and a small motor division distributed entirely through the mandibular branch of the nerve. The skin of the face and the mucous membrane of the mouth, tongue, and nose are supplied by pain, tactile, and thermal fibers which pass into all three branches of the trigeminal nerve; these are the ophthalmic, maxillary and mandibular nerves. The cornea differs from other parts of the face, in that only painful sensations may be elicited by stimulation of this region (Schäfer,¹ 1900). Sensory fibers also accompany the motor root into the brain stem, their unipolar cells of origin forming the mesencephalic nucleus, which is an unusual location for sensory cells. These fibers are believed to be proprioceptive and to supply

* Contribution No. 98 from the anatomic and physiologic laboratories, Northwestern University Medical School.

1. Schäfer, E. A.: Text-Book of Physiology, London, Longmans, Green & Co. 2:987, 1900.

the muscles innervated by the motor division of the trigeminal nerve (Willems,² 1911, Allen,³ 1919). The main sensory root carries the usual cutaneous sensations.

In cross section, the trigeminal nerve in the cat was seen to consist of fibers varying from unmyelinated fibers of 1.5 microns to the largest myelinated fibers of 16 microns. These latter belonged to the motor root and were always found on the dorsal surface of the nerve. There were very few of them, only 46 and 42 in two nerves counted,⁴ the majority of the motor fibers being between 10.8 and 11.7 microns in diameter. In two nerves 370 and 390 fibers of this size were found. Many of these, however, must be considered as sensory rather than motor since they occupied a more ventral position than any of the motor fibers.

The majority of the sensory fibers ranged from 5.3 to 8 microns in diameter and were scattered evenly throughout the sensory part of the nerve. In different specimens 2480 and 2459 of these fibers were counted. Intermediate fibers of 3.7 to 4 microns numbered 274 and 204 in the two different nerves, and fibers smaller than 3 microns appeared in equal numbers. In the pyridin silver preparations several bundles of unmyelinated fibers (392 and 400 in two different nerves) were seen scattered between the larger fibers. The small number of unmyelinated fibers in the trigeminal nerve is in marked contrast to the conditions in most other nerves, which contain numerous unmyelinated fibers (Kock,⁵ 1916).

In the cat the nerve is directed caudad and pierces the pons in the rostral ventrolateral region, and makes a slight curve dorsad (Fig. 1). The motor fibers separate almost immediately from the others and divide into several small bundles which pierce the pontine fibers and pass dorsad to the motor nucleus. The sensory division continues 0.5 to 1 millimeter caudad, and then breaks into a main sensory portion which passes dorsad to the main sensory nucleus, and a spinal tract which extends caudad into the medulla. Cajal,⁶ 1896, in a very com-

2. Willems, E.: Les noyaux masticateurs et mésencéphaliques du trijumeau, *Le Névrase* **12**:7, 1911.

3. Allen, W. F.: Application of the Marchi Method to the Study of the Radix Mesencephalica Trigemini in the Guinea-Pig, *J. Comp. Neurol.* **30**: 169, 1919.

4. The fiber counts of two or three different nerves are presented always in the same sequence—those from nerve A being given first, etc. The counts were made by enumerating the fibers in successive fields under the microscope, the stage being arranged to move in such manner that the lines of all series were parallel.

5. Kock, S. L.: The Structure of the Third, Fourth, Fifth, Sixth, Ninth, Eleventh and Twelfth Cranial Nerves, *J. Comp. Neurol.* **26**:541, 1916.

6. Cajal, S. R. y: Beitrag zum Studien der Medulla Oblongata, 1896.

prehensive description of the trigeminal nerve, claimed that the two sensory parts are formed from the sensory division by the splitting of each individual fiber in it, into a small branch passing to the main sensory nucleus and a larger branch passing to the spinal nucleus. In sections of brains of new-born rats and rabbits, prepared by the Golgi silver impregnation method, he was able to see this bifurcation. Since, with this method, all the fibers are never impregnated, it would be

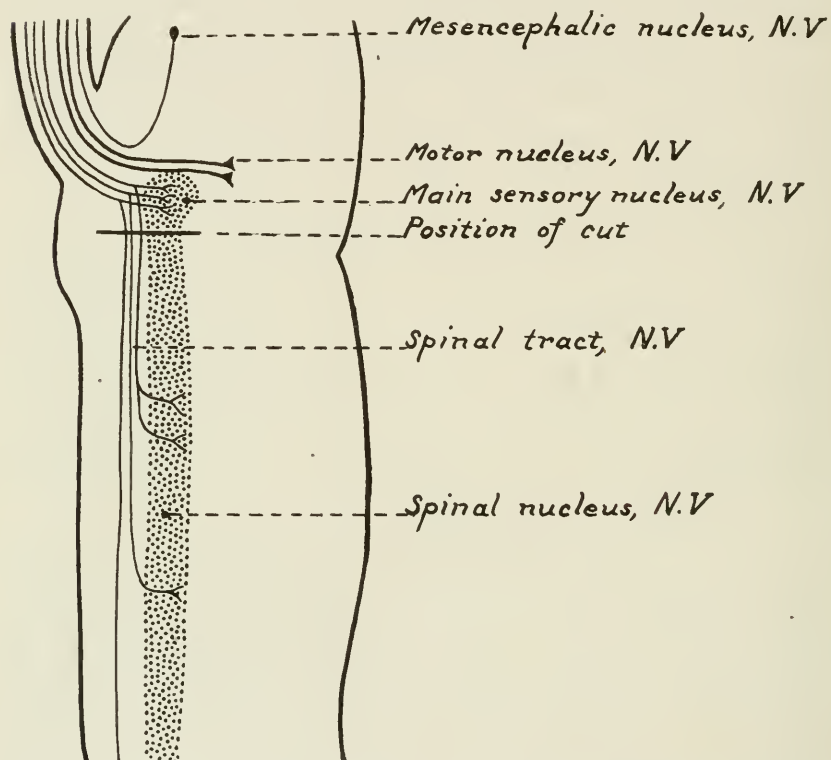


Fig. 1.—Diagram of a longitudinal section of the medulla, showing the central connections of the fifth nerve and the position of the experimental lesion.

impossible to determine whether or not every fiber bifurcates. Indeed, Kölliker,⁷ asserted that all the fibers do not do so, while Held,⁸ confirmed Cajal's description. That many of the fibers do bifurcate is evident in the pyridin silver preparations which we have examined, though it is not certain that all do. Actual bifurcations may be seen in

7. Kölliker, A.: Der feinere Bau des verlängerten Markes, *Anat. Anz.* **6**: 427, 1891.

8. Held, H.: Die Endungsweise der sensiblen Nerven im Gehirn, *Arch. f. Anat. u. Phys.*, 1892, p. 33.

many cases, and there is a noticeable increase in the total number of fibers as the nerve approaches the point of separation of the two divisions. This increase is mainly in the smaller fibers which are seen to pass to the main sensory nucleus. It may be noted that in no case are any of the unmyelinated, or the small myelinated, fibers seen to branch, indicating that these may, perhaps, pass entire into the spinal tract.

The descending or spinal tract forms, in the brain stem, a half-moon shaped bundle which caps the peripheral edge of the spinal trigeminal nucleus. In this bundle one can distinguish a superficial layer composed of vertical fibers only, and a deeper layer of vertical interlaced with horizontal fibers. The latter fibers are terminations and collaterals on their way to the spinal nucleus. Within the nucleus the fibers form a diffuse network between the individual cells. Kölliker,⁷ believed that, besides the endings in the substantia gelatinosa, there were collaterals which passed to the motor nuclei of the trigeminal, facial and hypoglossal nerves. Obersteiner,⁹ 1912, confirmed this observation, though Cajal⁶ 1896, had strongly denied it. In 1902, Breuer and Marburg¹⁰ described a bundle of fibers in the medial part of the tract, which they named "fibrae concomitantes", and which passed, in their opinion, through the spinal trigeminal nucleus to reach the nucleus solitarius, in which the tract terminated. Wallenberg,¹¹ 1904, again mentioned this bundle and suggested that it carried taste fibers from the mucous membrane of the mouth.

Cajal,⁶ in 1896, claimed to have traced the spinal fifth tract to a region just below the bifurcation of the pyramids. Wallenberg,¹² found in Marchi preparations from cats and rabbits whose fifth nerve had been cut, evidence of degeneration as low as the first cervical segment. Kljatschin,¹³ 1897, with a similar technic, followed the degeneration to the second cervical segment. Other investigators, Pomatowsky,¹⁴ in 1892, and Obersteiner⁹ in 1912, asserted that the tract extended to

9. Obersteiner, H.: Anleitung beim Studiren des Baues der nervösen Centralorgane in gesunden und kranken Zustände, Leipzig, 1912, p. 533.

10. Breuer, R., and Marburg, O.: Zur Klinik und Pathologie der apoplectiformen Bulbarparalyse, Arb. a. d. Neurol. Inst. a. d. Univ. Wien **9**:181, 1902.

11. Wallenberg, A.: Anatomischer Befund in einen als "Blutung in die rechte Brückenhälfte usw. aus dem Ram. Central. Arter. radicular N. facialis dextri" geschilderten Fälle, Deutsch. Ztschr. f. Nervenheilk. **27**:436, 1904.

12. Wallenberg, A.: Die secundäre Bahn des sensiblen Trigeminus, Anat. Anz. **12**:95, 1896.

13. Kljatschin, G.: Experimentelle Untersuchungen über den Ursprung des N. Trigeminus, Neurol. Centralbl. **16**:204, 1897.

14. Pomatowsky, A.: Ueber die Trigeminus-Wurtzel im Gehirne des Menschen, Arb. a. d. Inst. f. Anat. u. Phys. d. centr. Nervensyst., Wien **1**:98.

the second or third cervical segments. Gudden¹⁵ in 1892, after examining a calf with unilateral trigeminal aplasia, concluded that the spinal tract extended to the fifth cervical segment. Dejerine¹⁶ in 1914, considered the fourth cervical segment as the termination of the tract. In our preparations by the Pal-Weigert and pyridin silver methods, from cats in which one fifth nerve had been cut and allowed to degenerate, we found no evidence of degeneration below the upper level of the second cervical segment. This is in agreement with the evidence of Kljatschin, who based his conclusions upon Marchi preparations.

In examining the cross sections at various levels of the brain stem of the cat and of man, we found that in the lower levels of the tract there was a greater proportion of small myelinated fibers than in the upper levels, and at the lowest limits there was an actual increase in the number of unmyelinated fibers. As seen in cross section, the fibers of different sizes were evenly distributed, but were united into bundles of various sizes by the horizontal fibers, which had changed their direction to end in relation with the cells of the spinal fifth nucleus.

The largest fibers in the spinal fifth tract of the cat measured 9.9 to 10.1 microns in diameter. These were not very numerous at any point and gradually decreased in number as the tract descended. In two different brain-stems stained with the Pal-Weigert and pyridin silver methods, 149 and 157 fibers respectively were counted in the region of the superior olive. At the upper level of the inferior olive these fibers had decreased in number to 99 and 117, and a third preparation contained 129 in this region. At the lower level of the inferior olive 34, 15, and 27 were found, while at the decussation of the pyramids, there were 17, 8, and 14. In lower sections no fibers of this size were found.

Fibers of 5.3 and 8 microns in diameter were more numerous than the larger fibers and numbered 893 and 921 at the superior olive; 713, 718, and 697 at the upper level of the inferior olive; 612, 679, and 631 at the lower level of the inferior olive; and 312, 298, and 287 in the region of the decussation of the pyramids. All of these disappear above the upper level of the first cervical segment.

Fibers of 3.7 to 4.8 microns were found to be very numerous; 1102, and 1080 in the superior olivary region; 853, 839, and 872 at the upper level of the inferior olive; 704, 622, and 661 at the lower level of the olive; 417, 423, and 432 at the decussation of the pyramids; and 210, 197, and 184 in the upper region of the first cervical segment.

15. Gudden, H.: Beitrag zur Kenntniss der Wurtzeln des Trigemini Nerven, *Allg. Ztschr. f. Psych.* **48**:16, 1892.

16. Dejerine, J.: *Sémiologie des affections du système nerveux* **2**:800, 1914.

Myelinated fibers smaller than the last were equally numerous; 1161 and 1143 at the superior olive; 1109, 1134, and 1124 at the upper level of the inferior olive; 495, 472 and 392 at the decussation of the pyramids; and 149, 139 and 153 at the upper level of the first cervical segment.

The unmyelinated fibers in the pyridin silver preparations numbered 506 at the superior olive, 443 at the upper level of the inferior olive, 408 at its lower level, 392 at the decussation of the pyramids, and 502 at the upper level of the first cervical segment.

In the human brain-stem, the same relative proportion of the various sizes of fibers was found, although the fibers in each group averaged slightly larger than those of the cat; the largest being 11.3 microns instead of 10.1 microns.

To sum up this description, there is a decrease in the size and number of the fibers in the path as it descends; the larger fibers drop out entirely, as do some of the smaller ones, but many of the myelinated and unmyelinated fibers descend to the end of the tract. This indicates either that the larger fibers terminate in the upper levels of the tract or that, as a result of giving off collaterals on the way, they continue to the lower levels as smaller fibers. The actual increase in the number of unmyelinated fibers may be due to the loss of the myelin sheaths of the smaller fibers as they reach the lower levels, or, possibly, to the upward continuation into the spinal fifth tract of fibers from the tract of Lissauer, which is largely composed of unmyelinated fibers (Ranson,¹⁷ 1913). Since there is no line of demarcation between the tract of Lissauer in the upper cervical segment of the cord and the lower level of the spinal fifth tract, it is possible that those earlier observers who did not use Marchi technic confused these two fiber bundles, with a consequent disagreement on the actual boundary.

FUNCTIONAL DIVISIONS OF THE SPINAL TRIGEMINAL TRACT

The different branches of the fifth nerve seem to retain their identity within the spinal tract. Bregmann,¹⁸ 1892, after partially cutting the sensory root, observed a disturbance of painful sensibility in the ophthalmic division of the nerve and a degeneration in the ventral part of the tract. When the degeneration was in the dorsal part of the tract, sensibility in the maxillary and mandibular regions was involved. Obersteiner⁹ in 1912, described a similar distribution and further localized the fibers of the maxillary division in the medial part of the tract.

17. Ranson, S. W.: The Course within the Spinal Cord of the Nonmedullated Fibers of the Dorsal Roots: A Study of Lissauer's Tract in the Cat. *J. Comp. Neurol.* **23**:259, 1913.

18. Bregmann, E.: Ueber experimentelle aufsteigende Degeneration motorischen und sensiblen Hirnnerven, *Arb. a. d. Inst. f. Anat. u. Phys. d. centr. Nervensyst., Wien* **1**:73.

Other investigators have suggested that the different branches of the nerve also terminated at different levels of the tract. Solder¹⁹ in 1899, and Schlesinger²⁰ in 1902, believed that the frontal branch of the ophthalmic nerve extended the whole length of the spinal tract, the mandibular ended just above, the maxillary still higher, and the nasociliary branch of the ophthalmic extended only into the highest part of the tract. This arrangement was indicated in cases of syringomyelia, with a lesion proceeding rostrad through the medulla, in which the analgesia which resulted from the disease progressed in the form of narrowing circles from the periphery of the face, until only the nose and the medial side of the eye were left intact in the last stages of the disease. A case described by Woods²¹ in 1913, showed a similar area of analgesia surrounding the nose and eye.

Kutner and Kramer,²² 1907, and Wallenberg,¹¹ 1904, suggested a slightly different arrangement. According to them, the whole ophthalmic division extended to the lowest level of the tract, the maxillary not so far, and the mandibular division ended near the top. They also stated that the fibers from the lingual and buccal mucous membranes occupied the dorsal part of the spinal tract, while the fibers from the conjunctiva and the nasal mucosa were situated in the ventral part of the tract. In 1914, Dejerine,¹⁶ claimed that the area of analgesia centered around the nose and mouth.

In general, then, it may be said that the fibers supplying the most peripheral part of the face extend further down the spinal tract, while those supplying the central area terminate nearer to the beginning. It may also be said that those which extend further down the tract probably run ventrad to those which terminate at a higher level.

BLOOD SUPPLY FOR THE SPINAL FIFTH TRACT AND NUCLEUS

For the purpose of this investigation a consideration of the blood supply of the lateral area of the medulla, through which the spinal tract of the fifth nerve passes, is of prime importance. This area is supplied principally by the posterior inferior cerebellar artery. According to Stopford,²³ 1916, this is the largest branch of the vertebral

19. Von Solder, F.: Der segmentale Begrenzungstypus bei Hautanaesthesien am Kopfe, insbesondere in Fälle von Syringomyelie, *Jahrb. f. Psychiat.* **18**: 458, 1899.

20. Schlesinger, H.: Die Syringomyelie, 1902, p. 162.

21. Woods, A. H.: Segmental Distribution of the Spinal Root and Nucleus of the Trigeminal Nerve, *J. Nerv. & Ment. Dis.* **40**:91, 1913.

22. Kutner, R., and Kramer, F.: Sensibilitätsstörungen bei akuten und chronischen Bulbärerkrankungen, *Arch. f. Psychiat.* **42**:1002, 1907.

23. Stopford, J. S.: The Arteries of the Pons and Medulla Oblongata, *J. Anat. & Physiol.* **50**:131 and 254, 1916.

artery, and arises from its side at the lower end of the olive. "After curving round the lower border of the olive, it ascends in the neighborhood of the posterior lateral sulcus, usually posterior to the fila of the vagus and glossopharyngeal nerves, almost to the lower border of the pons, where it changes its direction and forms a loop with its convexity toward the pons. It now proceeds downwards, with a slight inclination toward the midline, on the restiform body and the other inferolateral boundaries of the fourth ventricle, to just below the calamus scriptorius, where it turns outward on to the vallecula to divide into its fully described lateral and medial branches, for the supply of the inferior surface of the cerebellum."

In the brain stem, Stopford found that the injected area was limited ventrally by the inferior olive, laterally by the tegmento-olivary tract, and medially by the fibers of the hypoglossal nerve. This area would therefore include the following structures:

1. Spino-thalamic tract.
2. Rubro-spinal tract.
3. Olivo-cerebellar fibers as they pass to the inferior cerebellar peduncle.
4. Dorsal vago-glossopharyngeal nucleus of the emerging fibers of the vagus and glossopharyngeal nerves.
5. Nucleus ambiguus.
6. Nucleus and tractus spinalis of the trigeminal nerve.
7. Ventral part of the inferior cerebellar peduncle.

We have found no record of experimental evidence which indicates a differentiation in the functions of the main sensory nucleus and the spinal nucleus. On the other hand, there have been many clinical cases recorded in the literature which suggest this possibility. Cases of occlusion of the posterior inferior cerebellar artery, strongly suggest that the pain and temperature fibers of the fifth cranial nerve terminate within the spinal nucleus of that nerve, while the fibers carrying tactile sensations terminate more rostrad in the main sensory nucleus. Cases of syringomyelia or of hemorrhage into the medulla which caused destruction of one or the other nucleus gave similar results. These cases have not as yet been collected, and the evidence which they offer has not been focused on the problem of sensory conduction in the trigeminal nerve. The investigation which I am here reporting represents an endeavor to assemble the clinical data and to secure experimental evidence on the question of such a differentiation.

II. CLINICAL DATA

SYMPTOM-COMPLEX OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

The symptoms which occur after occlusion of the posterior inferior cerebellar artery are remarkably constant and, in order to simplify the

presentation of the clinical evidence, they may be summarized in accordance with Gordinier,²⁴ 1911:

1. Sudden onset, usually without loss of, or disturbance in consciousness.
2. Diminution or loss of sensibility to pain and temperature in the area of distribution of the fifth nerve on the side of the lesion.
3. Diminution or loss of sensibility to pain and temperature in the limbs of the side opposite to the lesion, very rarely also, in the side of the face opposite the lesion. (This indicates an involvement of the secondary trigeminal path).
4. No paralysis or paresis of the muscles innervated by the trigeminal nerve.
5. Spontaneous pain or some form of paresthesia in the area of disturbed objective sensibility.
6. Tactile sensibility intact in all parts.
7. Sensibility of position usually intact.
8. Rarely a zone of intact sensibility in the region of the neck, between the area of disturbed sensibility in the face on the one side and that of the limbs on the other.
9. Tendency to fall or sway toward the side of the lesion, and for the head to drop forward toward the same side.
10. Bilateral nystagmus, more intense when the eyes are directed toward the side of the lesion.
11. Menière's syndrome of vertigo, revolving vertigo, auditory hallucinations, vomiting, and deafness on the side of the lesion.
12. Paralysis of the muscles of deglutition on the side of the lesion, with impaired sensibility of the pharynx.
13. Paralysis of the soft palate and larynx on the side of the lesion; vocal cords immobile in a cadaveric position; the voice hoarse and the speech merely a whisper.
14. The tongue, rarely, is a little weak on the side of the lesion, but the weakness usually soon passes away.
15. Taste may occasionally be lost on the anterior two-thirds of the tongue.
16. The facial and abducens nerves are not involved in the lesion.
17. Small pupil, ptosis, narrow palpebral fissure, and retraction of the eyeball on the side of the lesion.
18. Loss of sweating of the face on the side of, or on the side opposite to the lesion.
19. Persistent hiccough.
20. The pulse may be rapid (from paralysis of the vagus) or slow (from irritation of the vagus).

24. Gordinier, H. C.: Occlusion of the Posterior Inferior Cerebellar Artery, a Definite Symptom Complex, *Albany M. Ann.* **32**:585, 1911.

These symptoms are so typically constant, that they may be considered as a syndrome. For this reason, it will not be necessary to discuss the symptoms of the following cases at length, and those symptoms alone will be detailed which bear directly on the sensory defects in the trigeminal area.

Unfortunately, a clinical examination is not always as complete nor as carefully executed as one might desire, so that a number of the cases here reported lack adequate detail, and are of value only to confirm those which are more complete. For example, the clinician reports a "disturbance" in temperature or pain sensibility but does not state the manner of variation from normal. In some of the cases there was no mention of the integrity of certain sensibilities, and we must conclude that such sensibilities were not examined. In some cases diagnosed as "probable occlusion of the posterior inferior cerebellar artery," the sensory disturbances of the face and of the body were on the same side; this indicates involvement of the secondary paths of the fifth nerve rather than of the primary path. These cases, therefore, were either incorrectly diagnosed, or were so atypical that no conclusions on the distribution of the fibers of the fifth nerve may be drawn; they have consequently been omitted.

CLINICAL CASES OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

CASE 1.—The first case on record is that of Senator,²⁵ 1881. He tested for painful sensibility with a pin or needle prick, and with cotton wool for tactile sensibility. Tests for temperature were not recorded. Examination disclosed an area of total analgesia which included the left side of the forehead, nose, cheek, lips and chin, just to the midline; the sensibility to pain on the whole right side of the face remained intact. The tactile sensibility of the left side, on the contrary, was, when compared with the right side, little, if at all, impaired.

The necropsy disclosed a thrombus in the left vertebral artery, which extended into the posterior inferior cerebellar branch, and into the anterior spinal artery, and occluded them both. The resultant necrosis in the medulla was spindle shaped in cross-section and stretched from the middle of the olive to the lowest of the hypoglossal rootlets. It included among other structures (the restiform body, the border of the cuneate fasciculus, the motor nucleus of the vagus, part of the vagus fibers, part of the inferior olive, the accessory olivary nuclei, and the roots of the hypoglossal nerve), the spinal root of the trigeminal nerve, which was therefore destroyed, while the main sensory and motor nuclei of the trigeminal nerve, which were above the lesion, had remained intact.

CASE 2.—Another case was reported in 1881 by Remak.²⁶ The symptoms were typically those of occlusion of the posterior inferior cerebellar artery,

25. Senator, H.: Apoplektische Bulbärparalyse mit wechselständiger Empfindungslähmung, *Arch. f. Psychiat.* **11**:713, 1881.

26. Remak, R.: Krankenvorstellung eines Falles vom Hemianaesthesia alterans, *Berl. klin. Wchnschr.* **18**:300, 1881.

although there was no necropsy, and included, an analgesia on the right side of the face, which was particularly marked on the forehead and eyeball, and was accompanied by persistent spontaneous pain in the same regions. The activity of the motor fibers of the trigeminal nerve was entirely normal.

CASE 3.—In 1883, Senator²⁷ reported a case with necropsy. He tested for painful sensibility with a needle prick, but failed to mention his method of tactile examination. Temperature sensibility was not mentioned. The sensation of touch was normal on both sides of the face while needle prick on the left cheek, nose and inside of the nostril produced only tactile sensations.

At the necropsy a thrombus was found in the left vertebral artery. It occluded the lumen for a considerable distance, and also the opening of the posterior inferior cerebellar artery. The resulting lesion in the medulla spared the main sensory and motor nuclei of the trigeminal nerve, but included the descending or spinal root of this nerve, together with the vagal roots and nucleus, the restiform body, the fasciculus solitarius, the nucleus and roots of the hypoglossal nerve, and the cochlear nuclei and part of the olive.

CASE 4.—Wallenberg²⁸ announced the next case in 1895, though the necropsy report did not appear until 1901.²⁹ He did not detail his methods of testing the various sensibilities, but reported anesthesia to cold, painful and electrical cutaneous stimuli, on the whole of the left side of the face.

The necropsy in this case revealed an obturating thrombus in the left posterior inferior cerebellar artery. There was also a parietal thrombus in the right posterior inferior cerebellar artery, which only slightly narrowed the lumen. Microscopic examination of the medulla disclosed an anemic necrosis which included the ventral two-thirds of the spinal root and nucleus of the trigeminal nerve.

CASE 5.—Mann,³⁰ 1892, reported a case which he termed encephalomalacia medullae oblongatae. There was no necropsy to determine the etiology, but the symptoms were similar to those which follow occlusion of the posterior inferior cerebellar artery. An area of the right side of the face which extended from the vertex capitis to the chin and from the anterior edge of the ear to the midline, showed a marked reduction in pain, temperature and electrocutaneous sensibility and also a subjective burning pain. The mucous membrane of the right cheek also showed a reduction in painful sensibility. Tactile sensibility on the right side of the face, on the other hand, was entirely normal, as was all sensibility on the left side of the face.

CASE 6.—In 1897, Hun³¹ published an excellent, detailed discussion of a case which had come under his observation. He tested for painful sensibility with a pin prick, and for thermal sensibility with test tubes filled with hot or cold water. Examination of the patient revealed that the left side of the

27. Senator, H.: Zur Diagnostik der Herderkrankungen in der Brücke und dem verlängerten Mark, *Arch. f. Psychiat.* **14**:643, 1883.

28. Wallenberg, A.: Acute Bulbäraffection (Embolie der Art. Cerebellaris Post. Inf. Sinistra?), *Arch. f. Psychiat.* **27**:504, 1895.

29. Wallenberg, A.: Anatomischer Befund in einem als "akute Bulbäraffektion (Embolie der Art. Cerebellaris Post. Inf. Sinistra?)" beschriebenen Falle, *Arch. f. Psychiat.* **34**:923, 1901.

30. Mann, L.: Casuistischer Beitrag zur Lehre von central entstehenden Schmerz, *Berl. klin. Wchnschr.* **29**:244, 1892.

31. Hun, H.: Analgesia, Thermic Anaesthesia and Ataxia, *New York M. J.* **65**:613, 1897.

face and tongue, the inside of the left nostril, the right arm, right leg and right side of the body were entirely normal in tactile sensibility but were absolutely insensitive to pain and to differences in temperature. There were: decreased sweating on the left side of the face, a slight ptosis of the left eyelid, and, when the disease progressed, an appearance and gradual spread of left facial paralysis. All the muscles of mastication on both sides were spared which proved that the motor nucleus of the trigeminal was intact. Later, a partial sensitivity to painful stimuli returned in the body regions, but not on the face.

The examination of the brain at the necropsy was performed by Van Gieson, who reported that areas of softening existed on the left side, in the medulla, lower pons, and cerebellum, and that no lesions were found elsewhere. Histologic examination showed that the destruction involved the spinal tract and nucleus of the fifth nerve (as well as: the dorsal spinocerebellar tract; the column of Gowers; the fibers which pass between the cerebellum and inferior olive; part of the gray reticular formation; the motor nuclei of the glossopharyngeal and vagus nerves; and a very small part of the inferior olive), but did not include its main sensory and motor nuclei. The degeneration of the spinal fifth tract was complete, and extended to the upper part of the first cervical segment which Van Gieson claimed to be the lowest level of the tract.

CASE 7.—Wallenberg,³² 1901, reported a case which bore the earmarks of occlusion of the posterior inferior cerebellar artery, though the case did not come to necropsy (the patient was still living when the report was made). He found the usual hypalgesia and hypothermesthesia in the region supplied by all three branches of the right trigeminal nerve, which included the mucous membrane of the mouth and tongue. The areas of greatest disturbance were: the right temple, the right eye, the bridge of the nose, the slightest area about the angle of the jaw, and the ears and chin. Wallenberg mentioned a disturbance in tactile sensibility on the left side of the body, but made no reference to touch in relation to the face. It seems legitimate to conclude that, had there been any disturbance in tactile sensibility of the face, he would have mentioned it also.

CASES 8 AND 9.—In 1902, Breuer and Marburg¹⁰ reported two cases with necropsies. In the first one, the clinical examination revealed no disturbance on the right side of the face nor of the motor functions or tactile sensibility on the left. The region of the left side of the face supplied by the third branch of the fifth nerve, however, was insensitive to painful stimuli and was also the seat of a subjective pain.

At necropsy, a thrombus, which entirely occluded the opening of the posterior inferior cerebellar artery, was found in the left vertebral artery. Marchi preparations of the medulla showed areas of degeneration which included: the left spinal fifth tract below the level of the upper border of the decussation of the lemnisci; a bundle lateral to the posterior longitudinal bundle (the central fifth tract of Wallinger); and a bundle dorsal to the medial lemniscus (the ventral Haubenfeld of Spitzen, which is also a central fifth path).

The report of the second case was rather incomplete. There was no disturbance in motor activity of any part of the body, the sensibility to painful

32. Wallenberg, A.: *Klinische Beiträge zur Diagnostik akuter Herderkrankungen des verlängerten Markes und der Brücke*, Deutsch. Ztschr. f. Nervenheilk. 19:225, 1901.

and tactile stimuli was intact on both sides of the face and the right side of the face was also sensitive to thermal stimuli. But the left side of the face did not respond to cold.

The necropsy disclosed a thrombus in the left vertebral artery. The resultant lesion in the medulla involved a small part of the left trigeminal tract in its whole length; which particular part, was not specified.

CASE 10.—Rossolimo,³³ in 1903, described a patient (Case 5 of his report) whose symptoms were typically those of occlusion of the posterior inferior cerebellar artery, and though unconfirmed by necropsy, the case may be regarded as such. The patient showed marked disturbances of pain and temperature sensation in all areas innervated by branches of the left trigeminal nerve. In that portion of this area supplied by the maxillary division, there was also a subjective neuralgia. The right half of the body showed no disturbance in pain and temperature.

CASE 11.—In 1903, Mai³⁴ also reported a case of this type. An area of the right side of the face, reaching from the midline to a curve connecting the outer corner of the eye, the anterior part of the ear, and the corner of the mouth, and from the upper lip to the vertex capitis, was insensitive to pain and thermal stimuli. Tactile sensibility was absolutely normal over the whole body.

The necropsy disclosed a thrombus in the right vertebral artery, which partially occluded it and completely occluded the posterior inferior cerebellar artery. The resulting anemic necrosis in the right medulla included (besides a small part of the olive, the lateral nucleus, the nucleus ambiguus and the internal and external arcuate fibers), the ventral two-thirds of the spinal fifth tract and its nucleus.

CASES 12 AND 13.—Müller,³⁵ 1906, reported two cases which were perfectly typical, though there were no necropsies. In the first case, the left side of the face showed marked hypalgesia and hypothermesthesia. The disturbance was most marked in the region of distribution of the first branch of the fifth nerve, and did not include the mucous membrane of the mouth and tongue. All forms of sensations of the right side of the face were undisturbed, as was also tactile sensibility of the left side of the face.

In the second case there was a similar hypalgesia and hypothermesthesia which involved, however, the right side of the face. In this case, again, the most marked disturbances were in the region of distribution of the ophthalmic division of the fifth nerve. There was, in addition, a continuous subjective sensation of warmth on the right side of the face. Tactile sensibility was entirely intact on this side of the face and all sensibility was normal on the left side of the face.

CASE 14.—A case with fatal termination and necropsy twelve days after the onset of the symptoms, was reported in 1907 by Thomas,³⁶ before the American Neurological Association. From the onset, there was subjective pain and dis-

33. Rossolimo, G.: Thermoanästhesie und Analgesie als Symptomen von Herderkrankung des Hirnstammes, *Deutsch. Ztschr. f. Nervenh.* **23**:241, 1903.

34. Mai, E.: Ueber gekreuzte Lähmung des Kältsinnes, *Arch. f. Psychiat. u. Nervenh.* **38**:182, 1903.

35. Müller, E.: Ueber ein eigenartiges scheinbar typisches Symptomenbild bei apoplektiformer Bulbarlähmung, *Deutsch. Ztschr. f. Nervenh.* **31**:452, 1906.

36. Thomas, H. M.: Symptoms Following the Occlusion of the Posterior Inferior Cerebellar Artery, *J. Nerv. & Ment. Dis.* **34**:48, 1907.

turbance of pain and temperature sensibility on the right side of the face. This region was also completely dry, as the result of a complete adiadoresis which accompanied the other symptoms.

The necropsy disclosed a thrombus in the right vertebral artery, which extended into the posterior inferior cerebellar artery. Thomas did not find any marked microscopic lesions in the medulla, so could not describe the structures which were involved.

CASE 15.—In 1908, Mauss³⁷ reported a case which showed the symptoms typical of occlusion of the posterior inferior cerebellar artery. He had no necropsy to check his diagnosis. Painful sensibility of the whole left side of the face, particularly on the forehead and cheek, was markedly diminished. Hot and cold sensibilities were diminished in the whole of the left trigeminal area, whereas tactile sensibility in this region showed no variation from the normal.

CASE 16.—Wilson,³⁸ 1909, reported, before the Royal Society of London, a case showing the same typical syndrome. The symptoms were: loss of pain sensibility over all of the left side of the face and the left eyeball; painful stimuli, such as the prick of a pin, were felt as simple pressure; loss of cutaneous pain sensibility of the left side of the mouth, tongue, palate, fauces, uvula, and pharynx; loss of sensibility to all degrees of heat and cold on the left side of the face, and anhidrosis and vasomotor disturbances in the same region. Tactile sensibility and localization of touch were intact on both sides of the face, as was the motor control of the muscles innervated through the motor root of the fifth nerve.

CASE 17.—Harris,³⁹ 1910, also reported a case before the Royal Society of Medicine of London. The patient complained of a severe neuralgia of the left side of the face, which was particularly localized over the malar bone, above the left temple, and, at first, on the left side of the nose. Examination showed an analgesia and athermesthesia over the whole left side of the face, but no disturbance of tactile sensibility on either side. This case may be diagnosed as occlusion of the posterior inferior cerebellar artery notwithstanding the lack of a necropsy, as the typical syndrome which follows this lesion was observed.

CASE 18.—In the same year, 1910, Ludin⁴⁰ described a case of thrombosis of the left vertebral artery, which had come to necropsy. Examination disclosed an analgesia and athermesthesia of the left side of the face and a slight disturbance of pain and temperature sensibility on the right side of the forehead. Tactile sensibility over the whole body was normal, as was motor control (the author does not mention the muscles of mastication in particular, so they were undoubtedly capable of function).

The necropsy disclosed, in the left vertebral artery, a thrombus which occluded the opening of its posterior inferior cerebellar branch. Histologic

37. Mauss, T.: *Klinische Beiträge zur Diagnostik Bulbäre Herderkrankungen*, Deutsch. Ztschr. f. Nervenheilk. **34**:398, 1908.

38. Wilson, S. A. K.: *A Case of Thrombosis of the Left Posterior Inferior Cerebellar Artery*, Proc. Roy. Soc. Med., Neurol. Sect. **2**:52, Pt. 2, 1909.

39. Harris, W.: *A Case of Thrombosis of the Left Posterior Inferior Cerebellar Artery Followed by Severe Trigeminal Neuralgia in the Analgesic Facial Area*, Proc. Roy. Soc. Med., Neurol. Sec. **3**:81, Part 2, 1910.

40. Ludin, M.: *Ueber Thrombose der Arteria vertebralis*, Deutsch. Ztschr. f. Nervenheilk. **40**:380, 1910.

examination of the medulla revealed a lesion at the level of the middle third of the olive, which included: the formatio reticularis, the nucleus ambiguus, the spinocerebellar, spinotectal and spinothalamic tracts, a small part of the restiform body, the substantia gelatinosa Rolandi, and the spinal root of the fifth nerve.

The symptoms in this case were somewhat atypical, in that there was a disturbance in pain and temperature sensibilities on the side of the forehead opposite to the lesion. It may be noted that Ludin mentions only a "disturbance" of these sensibilities on the right, rather than a lack of them such as existed on the left side. It is possible that this disturbance was due to involvement of some secondary trigeminal fibers as they passed through the reticular formation from right to left, as the whole reticular formation was evidently involved in the lesion.

CASES 19, 20, AND 21.—In 1911, Gordinier²⁴ reported three cases which, from the clinical picture, he diagnosed as occlusion of the posterior inferior cerebellar artery, though no necropsies were obtained. The first case was very incomplete, mention being made only of tactile sensibility, which was normal upon both sides of the face. The second case showed complete loss of pain and temperature sensibilities over the whole of the left side of the face; the tactile sensibility of that side was entirely normal. The third case showed, in addition to a distinct reduction in pain and temperature sensibilities over the whole of the right trigeminal area, a marked anhidrosis of the right side of the face. Tactile sensibility was intact over the whole of the body.

CASE 22.—In the same year, 1911, Head and Holmes⁴¹ described a case which they called occlusion of the posterior inferior cerebellar artery. They used a prick of calibrated intensity to test pain sensibility, cotton wool to test tactile sensibility, and metal tubes (silver, 1.25 cm. in diameter) filled with water at various temperatures to test thermal sensibility. Examination revealed an insensibility to prick over the whole left side of the face. The threshold stimuli for pressure-pain, tactile, and thermal sensations were normal and equal on the two halves of the face. The patient complained, however, that all temperature seemed "duller" on the left side of the face than on the right.

CASE 23.—Courtney⁴² reported a case in 1912 which, from its typical syndrome, he believed to be due to thrombosis of the left posterior inferior cerebellar artery. Tactile sensibility was well preserved on the right side of the face, while pain was markedly blunted. Heat was perceived in part, as such, and cold, when felt at all, produced a sensation of warmth.

CASE 24.—Salmon,⁴³ 1912, reported a case of occlusion of the posterior inferior cerebellar artery with typical symptoms. He observed: complete insensibility to thermal, painful, and electric stimuli in the region of the face supplied by the second division of the right trigeminal nerve; slight diminution in these forms of sensibility in the remainder of the right side of the face; subjective neuralgia in the same region, accompanied by a subjective

41. Head, H., and Holmes, G.: Sensory Disturbances from Cerebral Lesions, *Brain* **34**:102, 1911.

42. Courtney, J. W.: A Case of Thrombosis of the Left Posterior Inferior Cerebellar Artery, *Boston M. & S. J.* **166**:329, 1912.

43. Salmon, D. A.: Di un Caso di Trombosi dell' arteria Vertebrale a della Cerebellare Posteriore ed Inferiore, *Sperimentale* **66**:442, 1912.

feeling of extreme cold or, as the patient described it, "petrification"; distinct anhidrosis on the right side of the face and hyperhidrosis on the left side. Tactile sensibility was absolutely normal on both sides of the face.

CASE 25.—The next year, 1913, Robinson⁴⁴ reported a typical case. He used the usual pin prick test for pain sensibility, and for tactile sensibility he used in addition to cotton wool, Von Frey's hairs. On the left side of the face, he found an absence of sweating, and a subjective feeling of numbness. Tactile sensibility was normal over the whole body except on the left cornea, from which tactile stimuli elicited no response. Painful sensibility was decreased over the whole of the left side of the face; and heat and cold sensations, though produced by equally weak stimuli on both sides, were much duller from the left than from the right side of the face. The control of the muscles innervated by the fifth nerve was intact.

CASE 26.—Cadwalader,⁴⁵ 1913, reported a case which he believed due to partial occlusion of the posterior inferior cerebellar artery by a thrombus. The patient showed a distinct diminution of heat, cold, and painful sensibilities within the region of distribution of the second division of the trigeminal nerve. The area involved was about the size of a fifty cent piece, and lay just over the malar bone. It did not extend forward to the nose, nor backward to the ear, nor was either cornea anesthetic. The nasal mucous membrane showed its normal sensibility, while that of the mouth and pharynx was almost insensitive. Tactile sensibility was unimpaired over both sides of the face.

CASES 27, 28, 29, 30, 31 AND 32.—In 1913, Goldstein and Baum⁴⁶ reported six cases of occlusion of the posterior inferior cerebellar artery, but gave, however, very meager data. In their first case, they reported disturbed sensibility on the right side of the face and tongue, but failed to mention either the type of sensibility which was involved, the nature of the disturbance, or the method they used to test for sensibility. Accompanying the sensory disturbance, there was a right sided conjunctivitis. The necropsy in this case revealed a degeneration of the spinal root of the right trigeminal nerve and of part of the substantia gelatinosa.

In the second case the threshold for painful stimuli on the right side was much higher than on the left, which was normal. They reported the right spinal fifth tract partially destroyed, but failed to locate the lesion more accurately.

The third case was more complete. The left conjunctival and corneal reflexes were absent, and painful sensibility on the left side of the nose, cheek and tongue was lacking. Tactile sensibility on the whole of the face was entirely normal. The necropsy disclosed a lesion in the spinal fifth tract, the exact location of which the authors did not specify.

Their fourth case showed a lessened sensibility to pain on the right side of the face, accompanied by loss of the corneal and conjunctival reflexes on the same side.

44. Robinson, G. W.: Occlusion of the Posterior Inferior Cerebellar Artery, *J. A. M. A.* **61**:179 (July 19) 1913.

45. Cadwalader, W. B.: Thrombosis of the Right Posterior Inferior Cerebellar Artery, *J. Nerv. & Ment. Dis.* **41**:375, 1913.

46. Goldstein, K., and Baum, H.: Klinische und anatomische Beiträge zur Lehre von Verstopfung der Arteria cerebelli posterior inferior, *Arch. f. Psychiat.* **52**:335, 1913.

In the fifth case, the sensibilities to temperature and pain of the left side of the face were entirely destroyed; corneal and conjunctival reflexes were partially lost on this side, and a subjective pain was felt in the same area.

The sixth case showed disturbance of pain and temperature sensibilities on the left side of the face and, on this same side, subjective painful and cold sensations.

In spite of their incompleteness, these cases point to a relationship between disturbance in pain and temperature sensibilities and destruction of the spinal fifth tract.

CASES 33 AND 34.—Two cases, in which the symptoms were very typical, but in which no necropsies were performed, were reported in 1914, by Gillis.⁴⁷ In the first case, there was absolute loss of pain and temperature sensibilities of the part of the face innervated by the first and second branches of the right trigeminal nerve, and a subjective numbness of the same region. There was ptosis of the lid, complete anesthesia of the cornea and conjunctiva, and enophthalmus of the eyeball on the right side; and the patient complained of a "peculiar feeling" in the right eye, and that his right nostril felt swollen. Tactile sensibility and tactile discrimination, as tested with cotton wool and a compass respectively, were found to be normal over the entire body.

The second case showed a complete analgesia and a hypothermesthesia of the upper part of the right side of the face, and an entire absence of corneal and conjunctival reflexes. Heat, warmth, and cold, when perceived at all, were accompanied by unpleasant affective elements. Tactile sensibility, as in the last case, was entirely normal.

CASE 35.—In the next year, 1915, an unusually good report of a case of occlusion of the posterior inferior cerebellar artery was published by Winkler.⁴⁸ He found painful sensibility destroyed and thermal sensibility much diminished (melting ice felt only cool, and hot objects, warm) throughout the whole of the left trigeminal area. The boundary line of this area was carefully determined. Anteriorly it corresponded with the median line, then passed over the top of the head, and returned in front of the ear and along the chin to again reach the median line. The left half of the oral cavity was analgesic, the tongue and palate being involved up to the midline; the left cornea was entirely anesthetic and no reflexes could be elicited from it nor from the conjunctiva. The palate and left side of the face were swollen; the latter was also markedly hyperemic and hot to the touch, but exhibited no trophic abnormalities. Winkler employed a plug of cotton wool, a small pencil and a pair of compasses to test tactile sensibility and localization, and found them diminished on both sides; but the motor control of all muscles innervated by the trigeminal nerve was entirely normal.

CASE 36.—In the same year, 1915, Spiller⁴⁹ described a case of occlusion of this same artery. The left side of the face was entirely insensitive to pain (pin prick), except over a small area below the mouth. Temperature sensibility was quite normal, though subjective "burning" in the region of distribution of the supraorbital nerve was noted. Tactile sensibility was intact

47. Gillis, A. C.: Occlusion of the Posterior Inferior Cerebellar Artery, *J. A. M. A.* **63**:1550 (Oct. 31) 1914.

48. Winkler, C.: A Case of Occlusion of the Arteria Cerebelli Posterior Inferior, *Proc. k. Acad. v. Wetensch. Amsterdam*, versl. **17**: Pt. 2, 914, 1915.

49. Spiller, W. G.: Remarks on the Central Representation of Sensation, *J. Nerv. & Ment. Dis.* **42**:399, 1915.

over the whole face. Spiller concluded from this case that there is a differentiation of pain from temperature fibers in the spinal tract of the fifth nerve.

CASE 37.—In 1919, a case was reported by Hausman.⁵⁰ He found analgesia of the entire left side of the face, and complete athermesthesia of the left temple, left cheek, and left side of the forehead, whereas tactile sensibility, tested with cotton wool, was normal over the whole of the face.

CASE 38.—Owen and Leighton,⁵¹ 1919, published the discussion of a slightly atypical case. They found that the right side of the face was insensitive to temperature stimuli; the face on the right, the inner cheek on the left, and the forehead, tongue, palate, and pharynx on both sides, were insensitive to pain. Tactile sensibility, tactile discrimination, and tickle sense were normal on both sides.

The bilateral disturbance on the forehead, and in the mouth and pharynx is obviously an aberrant condition, and may well be due to involvement of the secondary fibers of the fifth as they cross in the reticular formation from left to right (see Case 18).

CASE 39.—The last case published is that of Hall,⁵² 1920. The report was most incomplete, but the symptoms mentioned were quite typical. He wrote of a hemianesthesia on the right side of the face, but obviously included only pain and temperature sensibilities in this term, for he also stated that tactile and proprioceptive sensations were normal everywhere.

CASES WITH OTHER LESIONS

Besides cases of occlusion of the posterior inferior cerebellar artery, there have been other cases recorded in the literature, syringomyelia or hemorrhage directly into the brain substance, which have given symptoms of particular interest in the study of the central connections of the fifth nerve.

CASE 1.—The first case was reported by Wallenberg⁵¹ in 1904. The patient showed marked disturbance in painful sensibility and a lesser disturbance in that of temperature on the right side of the face, which included the bridge of the nose, the lips, and the mucous membrane of the mouth and tongue. Touch was normal over the whole of the face.

At the necropsy he found a lesion in the medulla which involved the right spinal fifth nucleus, a few cells in the main sensory nucleus, and the dorsal part of the spinal root. In this case, though a few cells in the main sensory nucleus were destroyed, tactile sensibility was intact. Probably the injury was so slight that the overlap in distribution of fibers masked any deficiency. The lesion in the spinal root and nucleus account for the disturbance in pain and temperature sensibilities.

50. Hausman, L.: A Case of Occlusion of the Posterior Inferior Cerebellar Artery with Cardiac Manifestations and Involvement of the Left Vagus Nucleus, *Arch. Neurol. & Psychiat.* **1**:145 (Feb.) 1919.

51. Owen, S. A., and Leighton, P. A.: A Case of Medullary Symptom-Complex Dependent on a Lesion of the Left Posterior Inferior Cerebellar Artery, *Lancet* **1**:1024 (June 14) 1919.

52. Hall, G. W.: Occlusion of the Right Posterior Inferior Cerebellar Artery, *Arch. Neurol. & Psychiat.* **3**:584, 1920.

CASE 2.—In 1906, Head and Thompson⁵³ reported a case of progressive syringomyelia. Examination showed analgesia and hypothermesthesia of the entire face. Heat was recognized a little more easily than cold, especially on the left side. The patient complained of a subjective "peculiar" sensation on the left side of the face, which felt as if it had been rubbed with menthol. Tactile sensibility (cotton wool), discrimination, and localization over the whole body were perfectly normal, as was the motor control of the muscles innervated by the fifth. (Only a motor disturbance of the fingers and hand is mentioned.)

The main sensory nucleus and the motor nucleus of the fifth nerve are so very close together that it seems probable that a lesion in one, marked enough to show a disturbance, would also involve the other. As the motor nucleus was not injured, it may be concluded, even in the absence of necropsy, that the main sensory nucleus was also spared.

CASES 3, 4 AND 5.—Three cases of syringomyelia were reported by Kutner and Kramer²² in 1907. The first case (Case 7 in their paper) showed a disturbance in the painful sensibility of the right side of the cheek and nose. Neither temperature nor touch sensibilities were disturbed, and the motor functions of the fifth were also entirely normal.

The lesion found at the necropsy included most of the spinal fifth nucleus, which showed considerable shrinkage, though it was not entirely gone. The upper part of the spinal and all of the main sensory nucleus were spared.

The second case (their Case 13) showed a disturbance of temperature and pain sensibilities in a semicircular area on the right side of the face, which extended from the forehead, halfway between the scalp and the eyebrows, to the middle of the chin. As the disease progressed, the boundary of the disturbed area advanced peripherally as a widening semicircle. At no time was there any disturbance of the motor fifth nerve.

The last case (their Case 14) showed a total loss of sensibility to both pain and temperature on the left side. The authors do not mention either tactile sensibility or motor fifth activity.

CASE 6.—Spiller,⁵⁴ in 1911, reported a case of syringomyelia which showed a complete loss of sensibility to pain, heat, and cold over the left side of the forehead, chin, and neck, and a partial loss over the left eyebrow and the cheek below the eye. The left side of the forehead also showed a slight diminution in tactile sensibility. There was, unfortunately, no necropsy in this case.

CASE 7.—Brun,⁵⁵ 1912, published a very complete report of a case. Tactile sensibility, as tested with delicate hairs and cotton wool, was intact over the whole face, and motor activity of the trigeminal was also entirely normal. Temperature and pain sensibilities, however, were greatly disturbed. On the right side: sensations of pain (needle prick) were absent from the whole face; warm

53. Head, H., and Thompson, T.: The Grouping of Afferent Impulses Within the Spinal Cord, *Brain* **29**:536, 1906.

54. Spiller, W. G.: Disturbances of Sensation in the Face in a Case of Syringomyelia, Indicating That the Upper Branch of the Trifacial Nerve Descends Lowest in the Medulla Oblongata, *J. Nerv. & Ment. Dis.* **38**:553, 1911.

55. Brun, R.: Ein Fall von doppelseitigen symmetrischen Erweichungssystem in verlangerten Mark nebst einem Herde im Kleinhirn, *Arb. a. d. Hirnanat. Inst. in Zürich von Monakow* **6**:273, 1912.

stimuli and cold stimuli could not be felt as such, although cold gave a "burning" pain on the upper third of the face, and on the lower two-thirds warmth was felt as cold; and reflexes from stimulation of the cornea, face, and mucous membrane were diminished. On the left side: there were athermesthesia and analgesia of the entire face except of the forehead and temple, and complete loss of the above mentioned reflexes.

The necropsy revealed, in the medulla, a syringomyelic cavity which occupied the area of the inner half of the substantia gelatinosa. The larger cells seemed to be involved in the degeneration, but the marginal cells were preserved. The degenerated region extended rostrad into the pons, and just reached the lower boundary of the main sensory nucleus; caudad, on the left side, it extended down to the cervical region, and the left spinal fifth tract was entirely degenerated; on the right side the dorsal part of the tract was spared, the central two-thirds alone showing a high grade degeneration.

CASE 8.—In 1915, an unusual case of multiple sclerosis was reported by Spiller.⁴⁹ The area of softening, indicated by complete paralysis of the muscles innervated by the motor fifth, was in the upper part of the pons, and involved the motor nucleus of this nerve. Tactile sensibility of the entire face was abolished, while pain sensibility was undisturbed and only a very slight disturbance in temperature sensibility was observed on the right side of the forehead. As the lesion included the motor nucleus, it is probable that the main sensory nucleus was also involved and, possibly, also the upper part of the spinal nucleus.

This case is especially interesting because it is the reverse of all the others. As, in them, tactile sensibility and motor function were spared together, so, in this case, they are alike involved, whereas pain and temperature sensibilities, usually destroyed, are here preserved. Correspondingly, the symptoms in this case were due to destruction of the motor and main sensory nuclei, with preservation of the spinal tract and nucleus, while the other cases all showed, primarily, lesions of the spinal structures alone.

SUMMARY AND DISCUSSION OF CLINICAL CASES

The evidence yielded by these cases, and the conclusions which seem justified by it, may be briefly summarized. Many cases, at necropsy, were found to have lesions of the spinal tract and nucleus of the fifth nerve on one or both sides. Such cases showed diminution or loss of pain and temperature sensibilities in areas on corresponding sides of the head, but never, unless the lesion actually included the main sensory nucleus, disturbance of tactile sensibility (Brun⁵⁵).

Other cases, which did not come to necropsy, showed the same dissociation of the tactile from the pain and temperature sensibilities of the head, with disturbances in the latter two; they also showed the other symptoms typical of occlusion of the posterior inferior cerebellar artery. As the region supplied by this artery extends rostrad, only to the lower border of the pons, the main sensory nucleus of the fifth nerve must have been spared in all these cases, the spinal nucleus and tract alone being involved.

In all these cases with sensory disturbances of pain and temperature but not of touch, additional evidence that the main sensory nucleus remained intact was found in the continuation of the normal function

of the motor fifth. The main sensory and the motor nuclei are so close together that they can be entirely distinguished only by differences in their cells, and, as shown by at least one case (Spiller⁴⁹), injury of the one would assuredly have been accompanied by injury of the other. As the motor nucleus must have remained intact in these cases, we may conclude that the main sensory nucleus was likewise uninjured.

One case is recorded which showed loss of tactile sensibility but not of pain and temperature sensibilities. Since in this case the masticatory muscles were paralysed, and no disturbance of medullary centers was indicated, it may be concluded that the main sensory nucleus, but not the spinal structures of the fifth nerve, was destroyed.

Thermal sensibility is disturbed in various ways. In many cases it is entirely abolished; in others only sensibility to heat is impaired, cold being felt in a normal manner; and in still others the application of heat elicited a sensation of cold, or cold a sensation of heat. The loss of heat sensibility but not of cold, in some cases, suggests a segregation of these fibers within the spinal fifth tract in the medulla.

Besides the objective symptoms, many patients reported subjective pain and temperature sensations in the anesthetic region. This may be attributed to a zone of hyperirritability surrounding the area of destruction. The frequent disturbance of sweat secretion in the areas of athermesthesia is of interest.

The most important conclusion which may be drawn from the above data is that the afferent impulses of pain and thermal sensibilities from the fifth nerve descend through the medulla in the spinal tract of that nerve, and pass at various levels to the cells of the spinal nucleus; whereas, the tactile impulses from the face pass to the cells of the main sensory nucleus. Such a distribution has been previously suggested by Winkler,⁴⁸ and Spiller.⁴⁹

III. EXPERIMENTAL WORK

The experiments to be described were carried out to test the validity, under more accurately controlled conditions, of the conclusion arrived at by a survey of the clinical evidence—that tactile impulses reached the main sensory nucleus of the fifth nerve, whereas thermal and painful impulses reached the spinal nucleus. We were able to cut the spinal root of the fifth nerve in the medulla at a level just below the bifurcation of the sensory fibers. This cut severed the whole of the spinal fifth tract from its peripheral portion, but left intact the fibers destined for the main sensory nucleus. The appearance or non-appearance of reflex activity in response to various stimuli, was the test used to determine which types of sensibility were impaired by the interruption of the spinal tract, and which types were left intact.

This method necessitated an exact knowledge of the sensory innervation of the face, especially of any overlapping of the two trigeminal areas at the median region, as a double sensory path would insure the presence of reflex activity even if one path were interrupted as described. To this end the whole fifth nerve on one side was completely severed proximal to the ganglion and the reflex activity from stimuli on both sides of the face was carefully tested.

A second objective of this research was the determination of the lowest level at which interruption of the spinal fifth root would still cause a dissociation of pain and tactile sensibility. For this purpose, we severed the root at various levels between the region of the origin of the tract and the region of the bifurcation of the pyramids, and again tested the integrity of the sensory paths by the presence or absence of a reflex involving them. Since cats are easier to work with than dogs, and also yield more delicate vasomotor reflexes, these animals were used throughout the course of the experiments.

SURGICAL TECHNIC

In the majority of the cases, urethane was used as the anesthetic, because reflex activity is not depressed, but rather facilitated, by this drug, and it was, therefore, particularly favorable for this work. An intraperitoneal injection of 0.75 gram of urethane (in solution) per kilogram of body weight was found to be satisfactory. In a few cases, when surgical anesthesia could not be produced by urethane alone, ether was used with it, and was given either by mask or by tracheal cannula. When reflexes were not to be tested till several days after the operation, the cats were anesthetised with ether alone, given by mask.

Entrance into the cranium and exposure of the spinal root of the fifth nerve was effected in two ways: (1) through the roof of the mouth, and (2) through the occipital bone. For the first approach, the cat was tied, dorsal side down, with the head just over the end of a board, the mouth was clamped open by a device arranged to keep it at its maximum opening, and the tongue was pulled out and tied. A cut was then made through the middle of the soft palate, and the two flaps, so formed, were held apart by threads which passed through each and were tied together over the bridge of the nose. This exposed the posterior wall of the nasopharynx. The mucous membrane was scraped with a chisel from the tympanic bulla and from the whole area of the basilar part of the occipital bone, laterad to the midline; then, with a dentist's drill, about three millimeters in diameter, the scraped medial wall of the tympanic bulla was removed. After removal of the bulla, which overhangs the occipital bone, it was possible to bore a hole in the latter just mediad to the bulla at its posterior third. The hole was made four to five millimeters in diameter and was

extended into the cranial cavity, where it usually opened into the inferior petrosal sinus. The resulting hemorrhage was allowed to drain into the mouth so that no intracranial clots, which would press upon the medulla, would be formed; the bleeding usually stopped in two or three minutes, when a small part of the medulla could be seen through the hole. This was the ventrolateral portion of the medulla at the level of the dorsal cochlear nucleus.

The spinal trigeminal fibers were cut with a curved cataract knife which had been ground so that only the distal centimeter possessed a cutting edge. This knife, held vertically with its cutting edge turned laterad, was passed through the hole in the bone and through the underlying medulla until its point reached the covering bone. The knife thus passed entirely through the medulla, from ventral to dorsal surfaces, just mediad to the fifth tract, and had its cutting edge directed towards that tract. The blade was then forced laterad by moving the handle in a medial direction, the hole in the bone serving as fulcrum; it was forced ventrad by raising the whole knife. The cut so made included the whole of the spinal fifth tract, a small area of the nucleus of the tract, and, occasionally, a small part of the restiform body and the overlying dorsal cochlear nucleus. The exact lesion produced was always checked by necropsy.

Occasionally the cat showed signs of shock after cutting the medulla. When apnea supervened, artificial respiration in the form of an interrupted air current was used.⁵⁶ The sensory tests were never made until the cat showed complete recovery from shock, so that the reflexes were as lively as in the normal animal.

When desired, blood pressure was taken from the carotid artery with a mercury manometer, and respiration was recorded from a tracheal cannula with attached tambour.

This method of severing the spinal tract proved to be most satisfactory when animals were to be experimented upon immediately. There was but little hemorrhage and only slight trauma to the tissues, and, therefore, less shock from the operation. It did not permit one, however, to make the cut accurately at a desired level, because the area exposed was small and the position of the medulla in the cranium slightly inconstant. For those experiments, therefore, in which it was essential to know the exact level of the cut, and desirable to make

56. It is interesting to note that very often diaphragmatic breathing alone reappeared, the thoracic muscles never regaining activity. It is possible that the afferent path for the respiratory reflex to the intercostals was interrupted. Coombs and Pike (*The Rôle of Afferent Impulses in the Control of Respiratory Movements*, *Proc. Soc. Exper. Biol. & Med.* **15**:55, 1918) believe that the impulse must ascend as far as the corpora quadrigemina before a reflex can result.

several cuts at different levels, the entrance into the cranium was made through the occipital bone in the following manner.

The hair was clipped from the dorsal surface of the head and neck, and the skin was cut from the vertex capitis down to the level of the first or second cervical vertebra. On the side on which the medulla was to be exposed, a cross cut was made mid-way between the ends of the longitudinal cut and was extended laterad to the ear. The two resulting flaps of skin were reflected and held out of the field by towel forceps. To eliminate hemorrhage as much as possible, a small amount of a 1:1000 solution of epinephrin hydrochloride was injected into the muscles along the line in which they were to be divided.

The muscles attached on either side of one lateral half of the inferior nuchal ridge were cut as near to the bone as possible; and also, by a second cut at right angles to the first, which extended rostrad as far as the superior nuchal line and caudad to the inferior edge of the first cervical vertebra, the muscles along the external occipital crest were divided. The bits of muscle which remained were scraped away from the underlying bone with a small periosteal elevator.

On the one side, then, the surface of the occipital bone was entirely clean. In this area, a circle of bone, with the superior nuchal line as a diameter, was ground very thin, by the use of the dentist's drill already mentioned, and the remaining sheet was removed with bone forceps. The preliminary grinding considerably reduced the amount of hemorrhage from the bone, and bone wax pressed into the open edges stopped the little which was unavoidable. After the circle of bone had been entirely removed, the dura mater covering the posterior part of the cerebellum and the lower and lateral part of the medulla could be seen. This dura was opened over the cerebellum to preclude any possibility of injuring the medulla. The cerebrospinal fluid was allowed to drain and the cerebellum was then lifted up and held backward, with a small retractor, in such a way that the whole lateral wall of the medulla could be seen. The spinal fifth tract was thus exposed along the whole of its course through the medulla and, with the knife already described, could be cut at any desired level without injury to adjacent structures.

This operation, in contrast to the other, involved much trauma and hemorrhage, and left the animal in a shocked condition. The muscles were, therefore, sewed back into place, the skin was sutured over the wound, and the cat was allowed to recover from the operation. Usually 20 to 50 c.c. of sterile isotonic saline solution was injected intravenously. From five to twenty-four hours after the operation the cat, as a rule, showed sufficient recovery to permit the continuation of the experiments.

Another method was used to cut the whole fifth nerve before it enters the pons (trigeminal neurectomy). This was always performed under strict asepsis and the cats were allowed to live for several days. The hair was thoroughly removed from the head anterior to the ear, and a skin incision was made just in front of the ear perpendicular to the main axis of the body. The temporal muscle thus exposed, was then cut and scraped from the underlying bone. A hole, 1.5 centimeters in diameter, was trephined through the frontal bone over the lateral edge of the frontal lobe of the hemisphere. This lobe was lifted with a small retractor, from the floor of the cranium, and the Gasserian ganglion in its depression on the apex of the petrous portion of the temporal bone was exposed. A cut was then made through the whole nerve proximal to the ganglion with a curved knife similar to that already described. The muscles and skin were sutured in the usual way and the cat was allowed several days in which to recover from the operation. It was found necessary after the operation to clean the eyes daily with mild protargin.

METHODS OF EXPERIMENTATION

At first, we attempted to test pain sensibility in different parts of the face by stimulation with needle electrodes which pierced the skin. This method was found to be very unsatisfactory; the cat repeatedly reacted to painful stimuli even after the spinal tract had been completely severed. The possibility suggested itself that deep pain fibers were present in branches of the seventh nerve which supply the muscles of expression, and that these afforded an afferent path for pain reflexes. In confirmation of this, we found that electrical stimulation on both sides of the face elicited reflexes in the cats with the entire fifth nerve cut on one side. It is interesting to note in this connection that Maloney and Kennedy,⁵⁷ 1911, demonstrated in man that deep sensibility of the face is retained after gasserectomy.

Since it was impossible satisfactorily to eliminate sensations carried by the seventh nerve, from this field, we chose to stimulate the cornea only. This is a particularly favorable region in which to test painful sensibility, as it is innervated exclusively by pain fibers from the fifth nerve (Schäfer¹). The nictitating membrane has a second sensory supply (either through overlapping of the opposite fifth, or from the seventh nerve), for stimulation of it called forth a response even with the entire fifth nerve on the same side cut. We were always careful, therefore, that the electrodes did not touch this membrane when we stimulated the cornea.

57. Maloney, W. J., and Kennedy, R. F.: The Sense of Pressure in the Face, Eye and Tongue, *Brain* **34**:1, 1911.

When painful sensibility of the cornea was intact, stimulation with a tetanizing current caused typical reflexes. The lids closed tightly over the eyeball; there was bodily struggling; the forepaws moved toward the eye; and the blood pressure usually rose markedly, and was accompanied by an irregularity in the rate of respiration.

The most delicate test for tactile sensibility mediated through the trigeminal nerve, is the sneeze reflex; very light stimuli in the interior of the nostril are sufficient to bring forth marked sneezing. A fine camels hair brush of nine to ten hairs, or a little cotton wool on the end of a probe, touched lightly to the distal part of the nostril until the sneezing commenced, was found effective. The cat under urethane, but not ether, was found to be just as sensitive to such stimuli as an unanesthetized cat. The sneeze was registered in the respiratory tracing.

As a third test for the integrity of sensation in the trigeminal area, we used the oculocardiac reflex. A very excellent discussion and review of the literature on this reflex was given by Naccarati⁵⁸ in 1921. The reflex, described first by Dagnini and by Aschner in 1908, consists of a slowing of the pulse, a modification of respiratory rhythm and, rarely, a lowering of blood pressure, as the consequence of pressure on the eyeball of the subject. It is exaggerated by severing the cervical cord (which interrupts the sympathetic connections with the medulla), and is abolished by cutting both vagi, so that the efferent path of this reflex must lie in the vagi. The afferent path is in the fifth nerve, for injury to that nerve prevents the reflex. The response, Naccarati believes, is probably not due to stimulation of pain fibers, for it is not produced by painful pressure upon the mandible or forehead.

Urethane augments reflex vagal activity (Becht⁵⁹), so that cats anesthetized by this drug are very favorable for this test. The reflex was elicited both by the usual pressure and by stimulation of the cornea with a tetanizing current, and the resultant slowing of the heart was recorded on the tracing from the carotid.

At the end of all experiments the cats were killed and necropsies were performed. The exact position of the cut was noted and the medulla was removed for histologic examination by the pyridin silver method of Ranson.⁶⁰ The tissue was imbedded in paraffin and cut in serial sections ten microns in thickness, in such a way that the fifth

58. Naccarati, S.: The Oculocardiac Reflex (Dagnini-Aschner Phenomenon), Its Use in Medicine and Psychology, *Arch. Neurol. & Psychiat.* **5**:40 (Jan.) 1921.

59. Becht, F. C.: Personal communication to the author, 1921.

60. Ranson, S. W.: The Pyridine Silver Method, with a Note on the Afferent Spinal Nonmedullated Nerve Fibers, *Rev. Neurol. & Psychiat.* **12**:467. 1914.

nerve could be followed into the medulla, the point of bifurcation of the fibers seen, and the whole spinal tract of the nerve below the level of the cut followed (Fig. 1). The whole of the lesion was always included in the material sectioned.

EXPERIMENTAL RESULTS

The sensitivity of the cornea to painful stimuli, that is, to a tetanizing current, was eliminated in each of the twenty-one cats in which the spinal root had been completely cut across. This analgesia appeared only on the side of the lesion, and did not involve the cornea

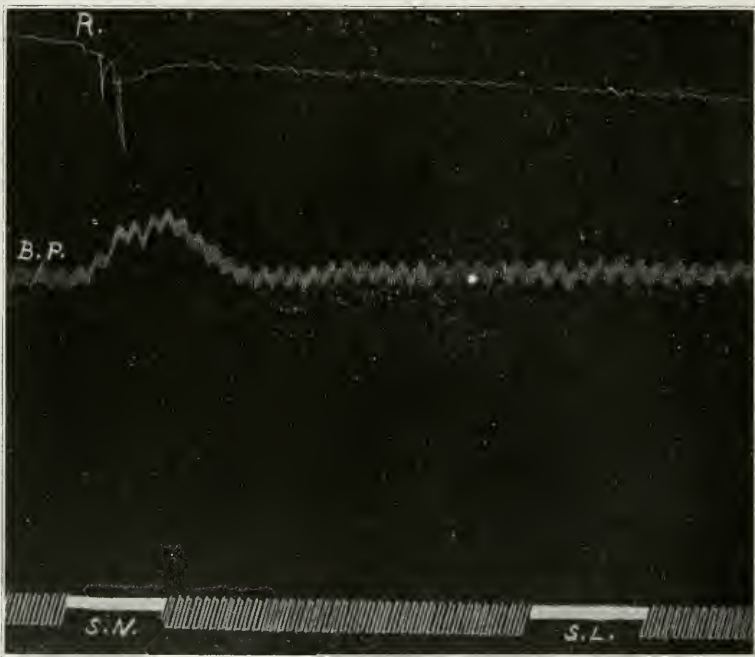


Fig. 2.—The vasomotor reflex from painful stimuli. *S N*, stimulation of the cornea on the normal side. *S L*, stimulation of the cornea on the side of the lesion. *R*, respiratory tracing. *B P*, blood pressure tracing.

of the normal side; stimulation of the latter always led to marked struggling, dilatation of both pupils, and a rise of 8 to 17 mm. in blood pressure. These are well recognized pain reflexes (Fig. 2).

The respiratory reflexes showed great variation. Stimulation of the analgesic cornea in no way modified respiration. Stimulation of the normal cornea caused: (1) no effect in four cats; (2) slight decrease in amplitude in three cats; (3) marked irregularity in seven cats; (4) apnea during stimulation in one cat; and (5) partial inhibition, followed by a period of marked irregularity, in one cat.

These respiratory variations were neither constant nor uniform enough to be considered as real indications of pain. The other reflex effects, struggling, dilatation of the pupils, and rise in blood pressure, were constant in their appearance, and may surely be interpreted as pain reactions. Since they were always present on the normal side and never on the side on which the spinal fifth tract had been cut, it seems reasonable to conclude that painful sensibility of the cornea was abolished by such a cut.

The sneeze reflex was used as a test for tactile sensibility on the last eleven cats. The touch of a little cotton wool in either nostril caused marked sneezing, preceded by licking movements of the tongue over the upper lip toward the nose. The reflex followed a latent period which varied from two or three seconds to seventy or seventy-five seconds and which was not constant even in one individual (Fig. 3). A rise in blood pressure usually accompanied the sneeze, but did not occur if the thorax was first opened; this indicated that the rise in blood pressure was secondary to the exaggerated inspiratory phase of respiration which precedes the sneeze. Cats with the spinal fifth tract of one side cut, and which were insensitive to painful stimuli of the cornea of that side, still gave a good sneeze reflex from tactile stimuli in either nostril. This was not due to an overlapping of fibers from the normal side, since four cats, with the whole sensory root of the trigeminal of one side cut, sneezed normally when stimulated with the camel hair brush on the normal side but did not sneeze or show any other indications of tactile sensibility when stimulated on the side on which the nerve had been cut. It may be noted that, in these cats, stimulation of either nostril with electrodes called forth marked pain reflexes. Either the pain fibers of the fifth nerve, in contrast to the tactile fibers, do overlap, or deep fibers from the seventh nerve were stimulated, or, possibly, the reaction was due merely to a spread of current, since the two nostrils are very close to each other.

Nor could the persistence of reflex effects from the nostril, after disappearance of those from the cornea, have resulted because the corneal fibers alone were included in the cut and on the assumption that the fibers which innervate the nostril terminate at a higher level of the spinal fifth tract than do those which innervate the cornea. In every case, except two, the tract was cut just below the bifurcation of the fibers, so that all the descending fibers must have been severed before any of them could have terminated. Only the fibers going to the main sensory nucleus were left, and the tactile sensations from the nose must, therefore, have passed along them.

Pressure on the eyeball of the normal side, in three cats under the influence of urethane only, caused distinct vagal slowing of the heart and also body struggling, while pressure on the eye of the side on

which the spinal fifth tract had been cut, in no case caused a response (Fig. 4). Electrical stimulation (tetanizing current) of the eyeball of the normal side, but not of the side which had been operated on, also led to slowing of the heart and to struggling movements. Since the tetanizing current, which certainly leads to painful sensations, produces the same effects as pressure, and since the responses to pressure stimuli

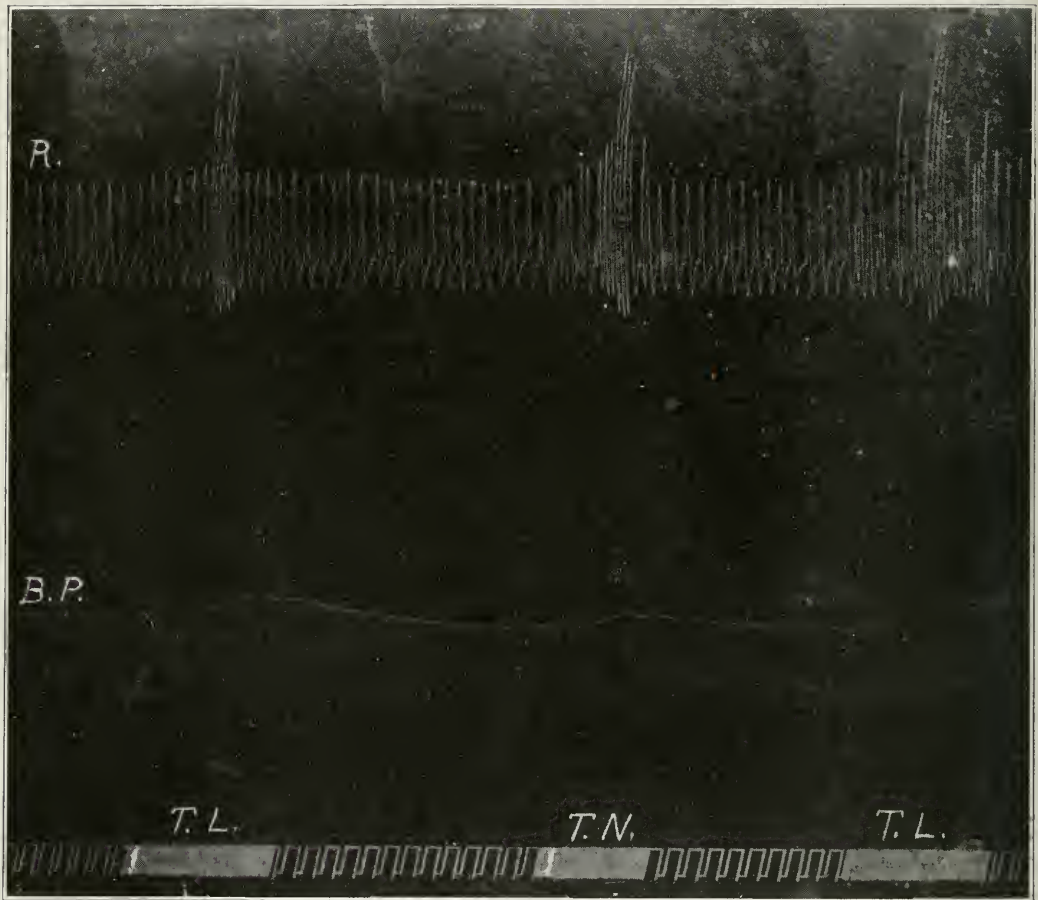


Fig. 3.—The sneezing reflex. *T L*, tickle of the nostril on the side of the lesion. *T N*, tickle of the nostril on the normal side. *R*, respiratory tracing. *B P*, blood pressure tracing.

are abolished by section of the spinal fifth tract, as are pain reactions, it seems that the oculocardiac reflex is due to stimulation of the pain fibers of the trigeminal nerve. Naccarati's observation that painful stimulation of the forehead and jaw does not slow the heart merely serves to emphasize the specific character of this reflex from the eye.

Histologic examination of the medullas showed that, in all cases in which the pain reflexes from the cornea had been entirely eliminated, the spinal fifth tract had been completely severed. In most, the spinal trigeminal nucleus and, occasionally, the reticular formation medial to the nucleus, were also slightly injured. The cut was usually found just below the bifurcation of the sensory fibers, but in two cases was about five millimeters below that point. The physiologic results in these two experiments were no different from the others. The cut was never found above the bifurcation of the sensory fibers, and the fibers going to the main sensory nucleus were intact in every case (Fig. 1).

In the preceding experiments, a high section of the spinal fifth tract was made, and histologic examination showed that the trigeminal fibers

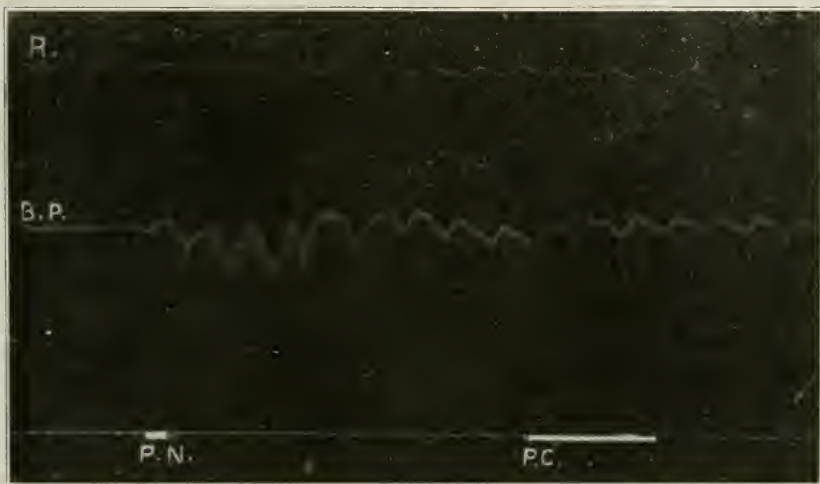


Fig. 4.—The oculocardiac reflex. *P N*, pressure on the eyeball of the normal side. *P C*, pressure on the eyeball of the side of the lesion. *R*, respiratory tracing. *B P*, blood pressure tracing.

had been cut just below their bifurcation, so that none of the descending fibers were left intact. To determine the level to which the pain fibers from the cornea descend before they terminate, a series of cuts in the tract were made, starting at the level of the obex, and gradually ascending the tract, until stimulation of the cornea no longer elicited reflexes. In four such cases, it was found that the cuts which eliminated the pain responses were 5.9, 6.7, 6.8 and 7.1 millimeters below the bifurcation of the fibers. In all except one case, the cut which proved effective was not more than 1.5 millimeters above the previous one (in the one exception, the effective cut was 2.1 millimeters above the previous one), so the margin of error was small.

These results indicate that the pain fibers from the cornea terminate just below the upper 5 to 7 mm. of the tract, and are in agreement with the clinical findings of Solder¹⁹ and Schlesinger,²⁰ to which reference has already been made—that the fibers supplying the eye and nose terminate in the upper part of the tract.

COMMENT

The experimental results are thus in entire accord with the clinical evidence, and demonstrate that painful sensations from the trigeminal area pass only by way of the spinal fifth tract and nucleus, while tactile sensations from the same region travel by way of the main sensory nucleus. Besides its diagnostic value, this fact possesses significance in two connections.

The spinal fifth tract is the direct upward continuation of the tract of Lissauer, and the spinal fifth nucleus is continuous with the substantia gelatinosa. Ranson⁶¹ has demonstrated that pain fibers from the spinal nerves pass into the tract of Lissauer and terminate in the substantia gelatinosa; so that the essential similarity of the painful innervation of spinal and cranial regions becomes apparent. The spinal fifth and Lissauer's tracts, and the spinal fifth nucleus and substantia gelatinosa are thus not only anatomically continuous but also physiologically identical.

Most, if not all, of the sensory fibers of the trigeminal nerve bifurcate and the smaller branch of each passes exclusively to the main sensory nucleus, while the larger reaches only the spinal nucleus. The dissociation of pain and temperature might possibly be explained as follows. It has not been conclusively demonstrated that all fibers bifurcate. Possibly the small myelinated and unmyelinated fibers carry pain and enter the spinal tract without bifurcation; the larger fibers carry touch (and possibly temperature—this case is not so clear) and send branches to both nuclei. Tactile sensation, having a double path, would thus persist after separation of the spinal tract from the nerve, while painful sensation could not.

This explanation is improbable for two reasons. First the vast majority of the trigeminal fibers certainly bifurcate; it is not probable that only the small fraction which may not do so is concerned in the transmission of pain sensations, while the two, hardly more important, sensations of touch and temperature are so generously supplied with transits. Secondly, if tactile impulses may follow either one of two paths, injury to the main sensory nucleus should not block them any more than does injury of the spinal nucleus, and if both of the nuclei

61. Ranson, S. W.: An Experimental Study of Lissauer's Tract and the Dorsal Roots, *J. Comp. Neurol.* **24**:531, 1914.

were involved, pain, as well as touch sensibility, should be lost. Yet in one case of syringomyelia (Spiller,⁶²) which involved the main sensory but not the spinal nucleus, pain was normal and touch was abolished. It appears, therefore, that pain impulses pass exclusively through descending fibers and touch impulses exclusively through ascending fibers. Since the ascending and descending fibers are, in most cases at least, branches of a single afferent fiber, it follows that both pain and touch impulses probably pass for a distance along the same nerve fiber.⁶²

As Lucas⁶³ points out, the character of a nerve impulse depends only on the condition of the nerve through which it is passing and not at all on its past history. If one impulse which reaches the point of bifurcation of a fiber follows only one branch, so must all others; and, as in the case of the trigeminal fibers, if some impulses reach each nucleus, it follows that all impulses must reach both. Yet impulses originating in pain end-organs can produce conscious and reflex effects only when they reach the spinal nucleus, and impulses originating in tactile end-organs are effective only when they reach the main sensory nucleus, though all impulses reach both. The following explanation seems plausible.

Forbes and Gregg⁶⁴ have secured evidence that a single stimulus of an end-organ may cause a series of nerve impulses in rapid succession to pass along the nerve fiber. The frequency of impulses may be different for each type of end-organ, and two different synapses may be "attuned"⁶⁵ to different frequencies; so that the impulse frequency from a painful stimulus may be able to pass the synapse to the spinal nucleus but not the one to the main sensory nucleus, while the impulse frequency from a tactile stimulus may be able to pass only the latter. Such a relationship would account for the observed facts, and is being further investigated.

62. Herrick, C. J.: *An Introduction to Neurology*, Philadelphia, W. B. Saunders Company, 1918.

63. Lucas, Keith: *The Conduction of the Nervous Impulse*, New York, Longmans, Green & Co., 1917.

64. Forbes, A., and Gregg, A.: *Electrical Studies in Mammalian Reflexes*, *Am. J. Physiol.* **39**:172, 1915.

65. Lucas (1917) suggests an "attunement" of this type. Immediately (0.002 sec.) after the passage of one impulse, a nerve fiber is entirely refractory to a second; later (0.013 sec.), a second impulse will be transmitted as one of low intensity; still later (0.03 sec.), as one of higher intensity than normal; and finally in the same manner as the first impulse was transmitted. The synapse is a region of conduction with a decrement, so that a series of impulses rapid enough to fall each in the relative refractory period of the last would be extinguished. One synapse might thus permit one frequency of sensory impulse to pass and bar a second, while another synapse, because of a different decrement, might act differently.

CONCLUSIONS

Besides offering data on the anatomy of the central trigeminal structures in cat and man, the following physiologic observations have been discussed:

1. Painful, and possibly thermal, stimuli in the trigeminal area produce conscious and reflex effects via the spinal fifth tract and nucleus only, while tactile stimuli produce effects via the main sensory fifth nucleus only (39 cases of occlusion of the posterior inferior cerebellar artery, 8 other human cases, and 21 cats with experimental lesions).

2. Pain fibers from the cornea terminate just below the upper seven millimeters of the spinal tract (cat). Pain fibers from adjacent regions descend further (human).

3. The fields of tactile sensibility of the two trigeminal nerves supplying the nose do not overlap (cat).

4. The adequate stimulus for the oculocardiac reflex is pain rather than pressure, and the reflex path includes the spinal fifth tract.

5. The spinal fifth tract and nucleus are functionally related to the tract of Lissauer and the substantia gelatinosa respectively.

6. Deep pain sensation from the facial muscles is probably carried by the facial nerve.

7. These results favor the view that impulses from more than one type of sensory stimulus may be transmitted by the same nerve fiber.

HEREDITARY AND NONHEREDITARY MENTAL DEFECT

A COMPARATIVE STUDY *

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The relation of hereditary influences to mental defect has been unquestionably established by the researches of students of both eugenics and feeble-mindedness. Little or nothing has been brought out, however, as to the *fundamental* inherited factors from which issue defective mentality. Furthermore, according to figures given by the various workers, the percentage of mental defectives having an impaired heredity ranges anywhere from 50 to 85. The authors are inclined to believe that an explanation for this variation of defective antecedents might be found in the geographical location or source of their material, as it is an experience common to all workers in mental deficiency that certain localities contribute an unusually large number of defectives with typical family histories, many members of whom are inter-related. It is our impression that approximately 50 per cent. of the patients at Letchworth Village are free from defective antecedents.

For years we have read, spoken and heard nothing but heredity, eugenics and segregation in relation to the defective. Strange to say, little attention has been given to the mental defective with a negative family history. It appeared to us that possibly a fruitful field might be presented if an effort were made to understand what differences, if any, could be found by an investigation of patients with hereditary and nonhereditary mental defect.

It is our aim to uncover certain leads, which will require much more intensive work before deductions of any value can be made. Conclusions based on the present study would be wholly unjustifiable for obvious reasons.

In selecting material for this study, cases were chosen in the order of the admission of the patients to an institution for mental defectives. Those cases whose histories were lacking, incomplete, or questionable as regards their validity, were immediately discarded. The cases selected were reviewed by three persons, all of whom were familiar with the histories.

* Read at the Interhospital Meeting at Hudson River State Hospital, March 16, 1922.

In this manner 100 cases were selected, half of which belonged to the group with hereditary mental defect and the other half to the group with nonhereditary mental defect. The hereditary group comprised patients one or more of whose ancestors had a definite history of mental defect. In order to qualify for the nonhereditary group, the family history of the patient had to be complete for three generations and free from all indications of defective, psychotic or other grossly maladaptive traits.

Each case was reviewed with special attention to etiology other than heredity, intelligence, physical characteristics, defects and diseases. Relative to etiologic factors other than impaired heredity, those assumed from the history given were classified as antenatal influences, accidents and injuries, disease of the central nervous system, and acute infectious diseases. The intelligence quotient or percentage of normal intelligence, rather than the actual mental age, was used to represent the intellectual development. This was necessary because the cases studied were of varying chronologic ages—some children and some adults. Physical defects and diseases were noted in terms of general medicine, neurology and endocrinology. The height and weight were used as an index of general physical growth and development.

HEREDITARY MENTAL DEFECT

Sex and Age.—In this group were twenty-nine males and twenty-one females. The ages varied from 5 to 30 years. Of these, thirty-nine were below the age of 16, and eleven above that age.

Etiology.—In forty-two cases there were no facts of an etiologic significance in the antenatal or developmental period of life.

Two patients gave a history of prolonged and difficult labor; however, there was nothing in their physical condition to point to this as having been a legitimate factor in causing their defective mentality.

At the age of 2 years, one patient had meningitis and another, poliomyelitis. Neither of these have any residuals of neurologic significance.

A cretin had poliomyelitis at the age of 4 and has now a characteristic extensor paralysis with a consequent pes equinus deformity of the right foot. Obviously the poliomyelitis does not account for her intellectual defect. One patient developed a hemiplegia at 13 months of age, and at the present time this is substantiated by the neurologic condition.

Relative to infectious disease, one patient had measles and pneumonia when 13 weeks old and another had whooping cough accompanied by convulsions during the first year of life. Neither of these patients have any physical evidence to show that a cerebral injury was produced at that time.

Intelligence.—The intelligence quotient ranged from 25 to 82, the average being 56. From the standpoint of classification, there were no idiots in this group; five were imbeciles, thirty-five were morons and ten were borderline types.

Physical Characteristics, Defects and Disease.—Height and Weight: The average height and weight of the males in this group was 56 inches (142.24 cm.) and 83 pounds (37.64 kg.), respectively. This represents an underheight of $2\frac{1}{2}$ inches (6.35 cm.) and an underweight of $10\frac{1}{2}$ pounds (4.76 kg.) in comparison with the normal average of the same ages.

Relative to the females, the average height and weight was $56\frac{1}{2}$ inches (143.5 cm.) and 91 pounds (41.27 kg.), respectively. This represents a deficiency of $2\frac{1}{2}$ inches (6.35 cm.) and $9\frac{1}{2}$ pounds in height and weight, respectively, when compared with the average normal females of the same ages.

Defects and Diseases: In twenty-four cases no signs which could be regarded as pathologic were found. Three patients showed a pigeon breasted deformity of the sternum with flared costal margins, indicating in all probability a rachitic condition during the developmental period. Two patients had the Hutchinson type of incisors, but in both of these the blood Wassermann test was negative. Five patients had pulmonary signs pointing to a probable tuberculosis. One patient had valvular heart disease. Fifteen patients had signs of an endocrinopathy. Eleven of these were of the status-thymico-lymphaticus type, two pituitary and two thyroid. Three had neurologic defects or disorders. In one of these the deep reflexes were absent, although this seemed to have no pathologic significance. Another had the characteristic residuals of an old hemiplegia. The third presented a unilateral retardation of development with muscular weakness, absent deep reflexes and double lateral nystagmus.

The blood pressure ranged from eighty (80) to 180 systolic, with an average of 108. The diastolic pressure varied between the lower and upper limits of 20 and 88, respectively, with an average of 69. Incidentally the average age of this group was $13\frac{1}{2}$ years.

Only two patients in this group had a positive Wassermann reaction in the blood. There was nothing in the general physical condition of either of these patients to cause one to be suspicious of a syphilitic infection.

NONHEREDITARY MENTAL DEFECT

Sex and Age.—Of this group, thirty were males and twenty were females. The ages ranged from 6 to 41 years. Twenty-seven of the patients were below the age of 16 and twenty-three above. The average age was 17 years.

Etiology.—Nineteen cases showed nothing of etiologic significance in their histories.

Three patients were born two months prematurely. There was nothing in their present physical condition to warrant the assumption that the prematurity was significant in the production of their mental defect.

Four patients had parents who were alcoholic, tuberculous or physically much below par. One of these patients, a mongolian idiot, was born of an eclamptic mother.

One patient gave a history of hereditary syphilis. This was not evident in his physical findings, however.

Ten patients gave a history of abnormal birth. Nine of these were instrumental deliveries after a prolonged labor and one a precipitate delivery; the latter was entirely negative from the standpoint of the physical examination. Of the other nine, five had neurologic defects: one had paralysis of the arm, one spastic paraplegia, two hemiplegia, and one progressive muscular atrophy.

Three patients had anterior poliomyelitis during the first two years of life; two of these now present the characteristic sequellae. Three had meningitis in early childhood. One of these is entirely deaf, one has hemiplegia, while the third shows no physical defects. One patient had a central lesion with convulsions during the first month of life and at present has choreiform movement of the hands and feet. One patient had sporadic convulsions during his entire infancy. This was a case of mongolian idiocy. One patient had had a spastic diplegia since birth. One patient had whooping cough during the first year of life, but the physical condition when examined was negative. One patient gave a history of a head injury followed by unconsciousness and convulsions, although physical examination failed to disclose anything of significance in this respect.

Two patients were undoubtedly not to be regarded as mentally deficient until the ages of 9 and 10, respectively. The former was a definite case of dyspituitarism, while the latter became defective following lethargic encephalitis.

Intelligence.—The intelligence quotient ranged from 10 to 91, the average being 41. Twelve patients were classified as idiots, twelve were imbeciles, twenty-four were morons and two were of the borderline type.

Physical Characteristic, Defect and Disease.—Height and Weight: The average height and weight of the males of this group was 57½ inches (146.05 cm.) and 92 pounds (41.73 kg.), respectively. This represented 1 inch (2.54 cm.) underheight and 1½ pounds (6.79 kg.) underweight. Relative to the females, the average height was 63

inches (160.02 cm.) and weight 122 pounds (55.33 kg.). This means an overheight of 4 inches (10.16 cm.) and an overweight of 22 pounds (9.97 kg.), as compared to the normal average for the same ages.

In twenty cases there were no physical defects or diseases.

Five patients showed a characteristic rachitic deformity of the thorax. Three patients had signs indicative of pulmonary tuberculosis. One patient presented a cardiac arrhythmia. One patient had a valvular heart lesion. One patient was extremely emaciated. Fourteen showed a defective internal glandular balance. Four of these were of the status-thymico-lymphaticus type, three of the pituitary, four of the thyroid, one of the gonadal, and two of the pluriglandular or mongolian type.

Eleven patients showed neurologic defects or diseases. One patient had an eighth nerve deafness, one monoplegia, three hemiplegia, two spastic diplegia. Two patients had the characteristic sequellae of anterior poliomyelitis; one had progressive muscular atrophy; one had marked choreiform and athetoid movements of the upper and lower extremities.

The systolic blood pressure for the group ranged from 86 to 130, the average being 116. The diastolic pressure ranged from 58 to 90, the average being 73. As stated, the average age of the patients in this group was 17 years.

A positive blood Wassermann reaction was present in only one case. This patient presented no other signs or symptoms indicative of a syphilitic infection.

SUMMARY

Two groups, one comprising fifty patients with a history of feeble-minded ancestors and the other composed of a like number of patients having an unimpaired heredity, were studied comparatively with respect to etiology, intelligence, physical growth and physical defects and diseases.

Etiology.—A history of possible etiologic significance other than heredity was found in eight patients belonging to the hereditary group and in thirty-one belonging to the nonhereditary group. In only one of the eight patients in the hereditary group does the physical condition warrant the assumption of the stated factor as a legitimate cause for the stunted intellectual growth.

In the nonhereditary group there was a direct relation between the physical condition as found at the present time and the so-called causative factor in fifteen of the thirty-one patients who gave a history of influences of etiologic significance. It would seem within reason to consider these fifteen patients as having a plausible explanation for their mental defectiveness.

It is to be noted that in all but three of these cases the assumed causative factor was of neurologic significance. The other three seemed to depend on an internal glandular imbalance.

Intelligence.—The distribution of intelligence for the two groups is of considerable interest. The average intelligence quotient of the hereditary group, fifty-six, was fifteen points higher than that of the nonhereditary group, which was forty-one. In the former group there were no idiots; five were imbeciles, thirty-five were morons and ten were borderline types. In the latter group there were twelve idiots, twelve imbeciles, twenty-four morons and only two belonging to the borderline type.

It may be added, generally speaking, that the patients with neurologic defects and diseases had a grade of intelligence corresponding to that of the idiot and lower imbecile group.

TABLE 1.—HEIGHT AND WEIGHT OF PATIENTS EXAMINED *

	Height, Inches	Weight, Pounds	Compared with Hereditary Group		Compared with Nonhereditary Group	
			Height, Inches	Weight, Pounds	Height, Inches	Weight, Pounds
Hereditary Group:						
Males.....	56.1	83.3			—1.6	—8.6
Females.....	56.6	91.0			—6.4	—31.0
Nonhereditary Group:						
Males.....	57.7	91.9	+1.6	+8.6		
Females.....	63.0	122.0	+6.4	+31.0		
Normal:						
Males.....	58.4	93.7	+2.3	+10.4	+0.7	+1.8
Females.....	58.9	100.4	+2.3	+9.4	—4.1	—21.6

TABLE 2.—PHYSICAL DEFECTS AND DISEASES

	Hereditary Cases	Nonhereditary Cases
None.....	24	20
Rickets.....	3	5
Hereditary syphilis.....	2	1
Pulmonary tuberculosis.....	5	3
Heart disease.....	1	2
Endocrinopathies.....	15	14
Neurologic diseases.....	3	11

Physical Growth and Development.—This is represented in a general way by the height and weight attained. The heights and weights of patients belonging to the hereditary group were consistently quite below those of the normal for the same ages, while the heights and weights of patients with a negative heredity were not appreciably below normal and in many cases exceeded those of the normal.

Relative to the average height, the males of the hereditary group were $2\frac{1}{2}$ inches shorter than the normal average for the same ages, while the males of the nonhereditary group were less than 1 inch shorter than the normal. The females of the former group were $2\frac{1}{2}$ inches shorter than the normal average, while those of the latter group were 4 inches above the normal average.

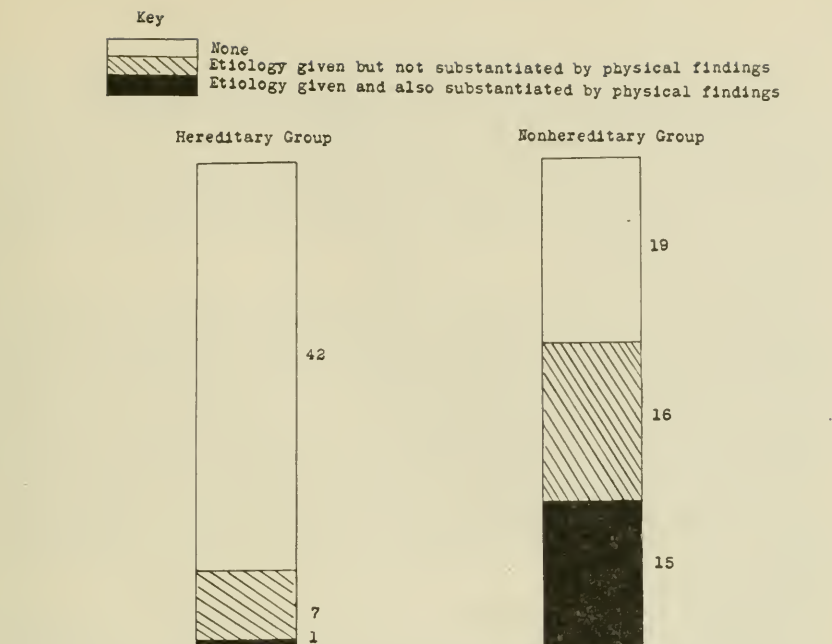


Chart 1.—Etiology of mental defect in the two groups studied.

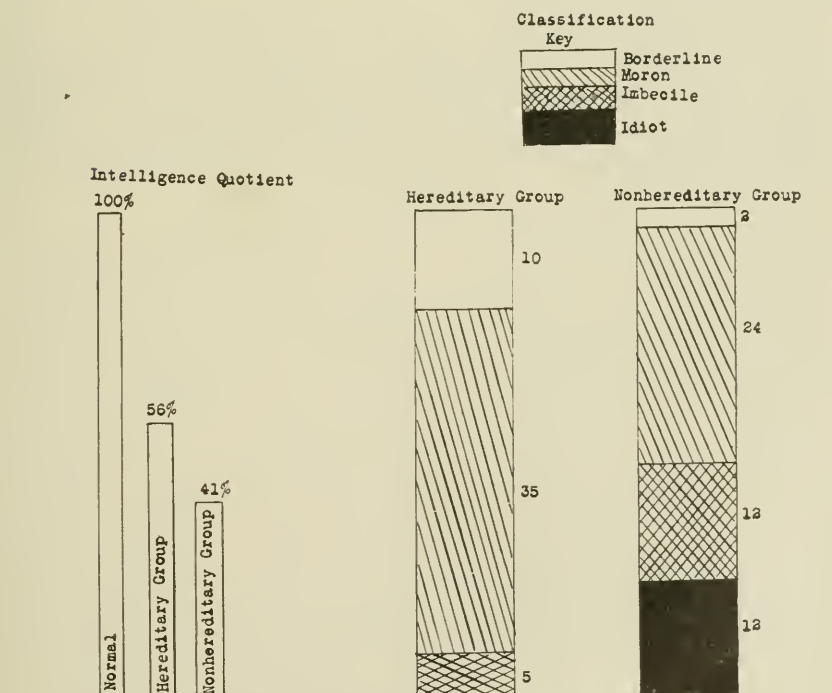


Chart 2.—Results of intelligence tests in patients studied.

As to the average weight, the males of the hereditary group were 10 pounds under the average normal weight for the same ages, while the average of the males of the nonhereditary group was only $11\frac{1}{2}$ pounds less than the normal average. The average weight of the females of the former group was $9\frac{1}{2}$ pounds less than the normal average, while the females of the latter group were 22 pounds above the average normal weight.

Physical Defects and Disease.—The whole number of the two respective groups showing physical defects and diseases did not differ materially, there being twenty-six cases among the hereditary group, and thirty among the nonhereditary group. On analysis, however, there were only three cases belonging to the former group showing a neurologic defect or disorder, while in the latter group were eleven cases.

One further salient feature is the fact that among 100 cases, over one half of them showed some physical disease or defect. Among this number were not included patients with minor defects of the eyes, ears, nose or throat.

This preliminary study would tend to indicate that there are decided differences between hereditary types of mental deficiency and those cases which for want of a better term might be called sporadic. Conclusions based on the foregoing findings would be unwarranted and quite unscientific.

It is planned to take up a more elaborate study along lines such as have been indicated in this paper of a number of cases sufficiently large to warrant conclusions. If this superficial study serves only to promote similar researches by other workers, one of its chief purposes has been accomplished.

A CASE OF AMBULATORY AUTOMATISM *

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This case is deemed worthy of report because of the remarkable history of fugues, which occupied almost five of the last fifteen years, in two attacks of which the patient married. Memory for the periods of automatism was recovered without hypnosis, although the patient repeatedly went into a hypnoidal state when the free association method was used. The following is an example of the replies received in this state. In association with the name Ac., which it was known had been adopted in earlier fugues, he said "Seems to be natural . . . another person altogether . . . the name seems right. . . I see him in a big store as a clerk. . . He used to drink . . . not to excess . . . worked all the time, too. . . The first time I met him in Colorado . . . stayed there a long time and went to Wyoming. . . There is some connection with Reid Murdoch.¹ . . . ("Think of Reid Murdoch.") There's a blank wall there . . . a basement with all kinds of labels. . . See all them cases I'm trying to read them . . . peaches, Monarch peaches . . . the first time they ever handled them. . . What makes me say that? . . . All you hear in that store is Ac. Ac., have you got this and have you got that, all the peas . . . sifted and extra sifted . . . Shreveport ginger ale. . . It begins to hurt . . . something seems to be pressing against my brain. . . ("Well?") I was going to say something but I don't know what it was, I don't remember." The following day he was again asked to think of Ac. and began talking of cases of grocery goods. He was asked, "Where does it seem to be?" "West some place . . . there's mountains all round it. . . ("Do you recognize them?") They're familiar but I don't know what the name is . . . lots and lots of sheep. . . ("What is the name on the store?") No name on the front of it . . . a concrete building . . . a big place . . . all kinds of automobiles going by . . . from all over the country . . . going to Yellowstone Park. . . ("The name of the street?") Yellowstone Highway. . . (His head rolled around, and he stared in front of him.) You know the guys told me to hold up my hands

* Read before the Chicago Neurological Society, Oct. 19, 1922.

1. A firm of wholesale grocers.

. . . that was in . . . Trinidad, Colorado." Later in this same conversation when asked how long he had been in Trinidad, he said, "I was always there" and gave his name as John Ac. He also said that the hold-up was "Yesterday, I guess . . . No, I'm home now, aint I?"

On waking from these states to full consciousness he remembered fully what had been said. It would take altogether too long to record the memories in the way they were recovered, and hence the material has been rearranged. No effort was made to recover the memories of some of the fugues and there are gaps in the last one.

REPORT OF CASE

History.—John P. Ab. was born in Chicago of Swedish parents, in 1881. The father had been a sailor before settling in Chicago and often talked to the patient of his travels. The family history was at first said to be negative, but in a dream analysis it was learned that a cousin of the father drank too much and often wandered away for a week or ten days at a time probably "to sober up rather than to wander like I do." A cousin of the mother also disappeared when the patient was 12, taking with him money that did not belong to him, and he has not since been heard from.

John was a sickly child until the age of 4. As a boy he was active, normally mischievous, fond of all games and always a leader. The home life was happy, though somewhat austere religious. Punishments were rare, the only whipping being for smoking cigarets at the age of 14. He remembered no dreams or night terrors and repeatedly asserted that he "was never afraid of anything." He was an average scholar in most subjects but "always at the top of the class in history." He completed the eighth grade at the age of 15, having been retarded by frequent change of residence. He recalled no heroes either in fact or fiction, but read with fascination everything he could find on travel and adventure. He did not care for the society of girls, but denied any feeling of bashfulness or discomfort when with them.

On leaving school he worked for his father in the fish business for three years and then became messenger in a bank. After about a year he had an attack of appendicitis for which an operation was performed. On recovering, attracted by the opportunity for travel, he enlisted in the United States Navy, in 1901. Here he was very happy, studied hard and was well thought of by officers and fellows. Before discharge he had been promoted to coxswain and had passed the examination for the grade of boatswain's mate.

In 1903, he developed tuberculous adenitis of the neck and during the next two years five or six operations were performed, a purulent discharge continuing throughout. The final operation was performed in 1905, with entire success. At this time, however, he had evidence of pulmonary infection with hemoptysis. He was retained in the navy for a few months as shipkeeper in a navy yard, and was then discharged with a pension of \$6 a month and a prophecy of death within three months.

According to his statements, he received as treatment for the tuberculosis, about a pint of whisky with cod liver oil and nux vomica daily. In addition, he secured elsewhere another pint of whisky so that for a long time he was taking about a quart of whisky daily. Prior to this illness he had hardly tasted whisky, though he drank beer occasionally. Throughout this period he

mingled little with women, always preferring the society of men. He denied, however, any feeling of bashfulness, and occasionally had relations with women. Venereal infection was denied.

The discharge from the navy was a bitter disappointment, and he felt that he was badly treated in the matter of pension and also in the operative work on his neck. He went first to Nebraska and Colorado but became worse. He then lived in Bovina, Texas, with a man to cook and care for him. He continued to drink whisky in large quantities for several months and was twice found drunk in the basement of the house. He then decided to give it up entirely, and though there was a severe craving, succeeded in doing so. His health steadily improved, and in the following summer, 1906, he weighed 160 pounds (72.5 kg.). He made many friends in Bovina and the neighboring ranches. During this summer he injured his urethra while riding a bucking broncho. Sounds were passed and a false passage established which gave him much trouble during the following seven or eight years.

Two months after the accident there was an exacerbation of the tuberculosis, with high fever and hemoptysis, and he was much upset by the assertion of the physician that an operation would be necessary on the other side of his neck. He, however, improved without operation, and gradually resumed a more active life. About one month after getting out of bed, he suddenly left home one night on his first fugue and came to himself two or three days later in a railroad wreck on the Santa Fé at Attica, Kan. For a long time no memories in connection with this episode could be revived. During the study he repeatedly told stories, remembered during the night, of occasions when he had seen persons killed. These memories had not been recalled in many years. Finally, it was learned that just one week before the fugue a man was shot down close beside him, and, much shocked, he drove home as fast as he could. On the day of the fugue he was on the prairie shooting and accidentally shot the friend who was with him, causing him to lose consciousness but not seriously injuring him. He was much frightened and drove furiously about 20 miles to a physician. On arriving, he was so exhausted that the physician gave him a drink of whisky, the first he had drunk in many months. That same night he left home, went to a neighboring town and drank heavily. He did not know where he was going when the train was wrecked.

Following this episode he returned to Bovina and rapidly improved in health. He then started to work for his brothers in a real estate business in Texas and Kansas. He became an excellent salesman and was very popular everywhere. But he began to have periodic spells of drinking which would start suddenly, usually preceded by a "blinding headache." They lasted from a day to a week or more, and then he would not drink again perhaps for months. In them it is not clear that he really lost himself prior to the next incident.

Early in July, 1907, he returned to the Chicago office from a successful selling trip. On the train he had a severe headache. On arrival in Chicago on a Friday morning he had breakfast and went to the office, where he turned in his money and settled his accounts. Feeling sick, he went to his mother's home in the afternoon and lay down. He knew nothing from then until the following Tuesday morning when he awoke in a hotel in Chicago. With him was a woman, and after some questioning he learned that he had married her at Crown Point (a nearby town) on the preceding Friday evening. He had known the woman for about six months and had occasionally

taken her to shows when he was in town. She was entirely respectable and he liked her, but he had never had any thought of marrying her, and he was much horrified at what had happened.

Later he remembered that he had been drinking heavily on the way home, having spent the whole day Thursday drinking in St. Louis. On reaching Chicago he had a drink before breakfast and several more during the morning, though he was not drunk. He also recalled that he had telephoned to the girl and made an appointment with her for the evening.

Following the discovery of his marriage he accepted the situation with apparent resignation and attributed it to drink. He introduced his wife to his relatives, but continued to drink, and three weeks later was taken to a hospital with "delirium tremens." On recovering, he went to his mother's home and to bed. This was in August, 1907. The next thing he remembered was finding himself in a hospital in Topeka, Kan., with "malaria," about Christmas, 1907. He was told that he had been found in a delirious state at the depot and taken to the hospital. Following this fugue, which was not studied further, he returned to Bovina and resumed work for his brothers, but did not attempt to see his wife. He stayed there for about a year and then, at the instigation of his eldest brother, visited his wife, and they agreed to live together. He furnished a home in a small town in Texas where they lived for about a year (1909), during which a son was born. The relationship was not congenial, and he had several drinking spells, in some of which he acted with wild extravagance and abandon. In 1910, his wife had an attack of typhoid fever, and the baby was sick. When convalescent she visited relatives in Chicago, and then refused to return to Texas. Finally, she moved away, and he lost track of her altogether.

For a time Ab. continued to work in Texas but, following his father's funeral in Chicago, he suddenly disappeared again about March, 1911, and came to himself two months later in New York City. He wired to his brother who came for him, and they stayed there for about a week, when John again disappeared. He was gone nine months; he came to himself in a small town in North Carolina, where he found that he had been working for practically the whole period of the absence, under his own name, as foreman of a construction gang in the building of a dam. He learned that he had done good work and was well liked, but had been drinking "something scandalous."

He returned to work for his brothers and had another short "absence" during 1912. In February, 1913, while in Mercedes, Texas, he received a telegram calling him to Chicago on account of the serious illness of his eldest brother, to whom he was deeply attached. He started at once, caught a train at Harlingen, but left it during the night somewhere before reaching Houston. He came partially to himself two or three months later in a hospital in Pine Bluff, Ark., again with "malaria." It was later learned that after leaving the train he had gone to Dallas and from there to Texarcana, where he opened an office for real estate work. When he left the hospital at Pine Bluff he was very much puzzled and could not entirely remember his past, but he found in his pocket letters addressed to himself at Texarcana. He therefore went there, and, finding himself accepted without question, carried on the work. About two weeks later, while inspecting some timber, he suddenly had a vivid "vision" in which he saw his "brother's funeral." This was so real and affected him so strongly that he drove 16 miles into town and telegraphed home asking about his brother. On receiving word that the brother was dead and buried, the memory of the earlier message

returned, and he proceeded at once to Chicago. It is of interest to note that during this fugue he had gone to Pine Bluff on business, and while there he had been introduced to a woman whom he married about eighteen months later.

After visiting Chicago, where he had some trouble with his brothers about financial matters, he went to a ranch in West Texas to recuperate, where he remained for about nine months. The trouble with his brothers then became more acute, and he decided to go into business for himself. He went to Pine Bluff, which he had frequently visited before, and after working for several months in saw mills, finally purchased one. During this time, early in 1914, he again met, accidentally, the woman he was to marry, at once recognized her and remembered her name, although his previous acquaintance with her was during a fugue. In June, 1914, he secured a divorce from his first wife, and in September married again.

Three weeks after the marriage, he was injured at the mill, several ribs being broken. This kept him from work, and he felt miserable and ill. Three weeks after the injury he disappeared from the hotel where he was living and came to himself ten days later in a hospital in Birmingham, Ala., where he had been registered for nine days under the name of John P. Ac. This was the first occasion on which, so far as he knew, he had ever assumed another name, although the initials were the same as his own. According to his story, he was paralyzed when he came to himself, but rapidly regained the use of his legs. The account received from the hospital stated that there was no record of any paralysis, but that he was admitted on Nov. 27, 1914, complaining of severe pain in the back and loins, so severe that he was "unable to walk." He had a traumatic, urethral stricture with retention of urine, and internal urethrotomy was performed. Later it was found that the right ureter was obstructed and there was great swelling of the right kidney, a further operation was performed in which the kidney was decapsulated.

It is worthy of note that though he remained in the hospital from Nov. 27 to Feb. 14, 1915, his wife of a few weeks did not once visit him, and on leaving the hospital he went to Florida for several weeks without seeing her. He said, however, that their relations were of the best, and he heard from her regularly.

On leaving Florida he rejoined his wife in Pine Bluff; shortly afterward they moved to Louisiana and lived with his wife's parents. Here he grew stronger and again started in real estate work. He was not happy, however, and complained that his wife's relatives sponged on him and spent all he could earn. A daughter was born in October, 1916. They stayed until the Pershing expedition into Mexico when John secured a position in the Materials Inspection Corps and was sent to El Paso. Here his wife joined him after a few months, and later they were transferred to Deming, N. M., where they remained until the corps was disbanded in 1917. On the entrance of the United States into the World War, John tried to enlist but was rejected. He secured a position as lumber inspector in camp construction work and was sent to Nashville, Tenn., in October, 1917. Here he rented a home and drove out daily to the camp, about 20 miles away. He asserted that he was happy here but found his work hard and the responsibilities, especially in the hiring and supervision of a number of men, very irksome. He felt, however, that it was his duty to continue with it. A second baby was born in March, 1918. In July, the home was quarantined for measles, and then his wife developed appendicitis for which operation was necessary. One morning in

August, shortly after the return of his wife from the hospital, he started for work as usual at about 5:30. He had trouble with the carburetor of his car and twice got out to adjust it. From then he remembered nothing until Oct. 25, 1918, when he came to himself on Boston Common with a bundle of laundry under his arm. He felt dazed and very ill, appealed to the police for help, after inquiring as to his whereabouts, and was taken to the psychopathic hospital. The records of that hospital state that no disease was found; he was an attractive person, and his mental condition appeared normal except for a specific amnesia for the preceding two months. He also gave a good history of his life with amnesic periods as outlined in the foregoing. No success was achieved in penetrating these periods, and efforts at hypnosis were unsuccessful.

The first contact with the memories of this period came accidentally in connection with a discussion as to the date of enforcement of prohibition. The patient, without hesitation, said that he was in Wilmington, Del., at that time and then said "But I have never been in Wilmington!" Later in a hypnoidal state the following account was obtained: On the morning following the trouble with his car in Nashville, he was in Louisville, Ky.; he remembered coming from the station but not anything before that. He was without a coat, his shirt was torn and his trousers were very dirty, so that a man he spoke to remarked that he "must have been on a hell of a tear last night to get in that shape." He had \$380 in \$20 bills and \$5 or \$6 in smaller change in his pockets. When he left home in Nashville, he had \$400 in \$20 bills with which he proposed to pay a debt. A bandage, which looked as if it "must have been applied by a doctor or a nurse," was on his left arm over a long cut. He had severe pain in his chest, which hurt when he breathed, and he could not lift anything. When he went to a physician a few days later, it was found that he had some broken ribs. On returning to Nashville, after leaving Boston, he learned that his car had been picked up from a ditch at the side of the road about 3 miles from the city; the hind end was "all stove in," and one wheel was smashed. Taken together, these suggest that the patient had been in an accident, though he has no recollection of it at all and does not know how he got to the station or to Louisville.

At Louisville he secured work for the government under the name Ac. as an assistant in building inspection. He was sent first to Norfolk, Va., and then in succession to Newport News, Wilmington, New York, New Haven and Boston. He recalled where he had worked and stayed in each place. On reaching Boston, he was feeling sick, went to a hotel and lay down for a time. The next event remembered was coming to himself on the Common.

After a stay of a few weeks in Boston he returned to Nashville but did not work for three or four months. He then secured a position in Michigan, where he stayed for about a year. He did not like the routine life he had to lead and was anxious to get into business for himself; he therefore resigned and again went to live with his wife's folks in Louisiana. Here he started buying and selling oil leases and had some financial disappointment due, he said, to "trusting people" too much. In the fall of 1919, he heard that his mother in Chicago was not well and came to visit her bringing with him his elder child. He stayed a few days and then returned to Louisiana. The child became ill on the train, and this worried him a great deal. Shortly after reaching home he went to Shreveport in connection with an oil deal in which he felt he had been cheated, and from that time remembered noth-

ing until June 10 or 12, 1922, a period of more than two and a half years, when he came to himself lying in a box car at Des Moines, N. M., with his head cut open and his pockets turned inside out. As a matter of fact he had been told by his brother, who had learned from the wife, that the family had moved from Louisiana to Houston, Texas, and had lived there about three months before he actually disappeared, but of this John had no recollection. It is also of interest to learn that, to his surprise, when he came to himself he was wearing the ring and button of a secret order without the least knowledge as to where he had acquired them.

At Des Moines he felt hazy; the world seemed strange, and he was afraid to speak to people lest they think him insane. He slept a great deal and was ravenously hungry when awake. He spent the first three days at a ranch, and then started to walk and work his way to Chicago, taking advantage of rides proffered by passing motorists. It was not until he reached Wichita, Kan., where he had once spent some months, more than a week later, that he fully realized who he was. In passing through Kansas City he happened to notice the hall of the secret order whose insignia he was wearing. Moved by impulse, he went in to find that he knew the whole ritual. Even this failed to bring back any recollection of where he had learned it. He reached Chicago about six weeks after coming to himself, and went at once to see Dr. Edward Ochsner, who kindly referred him to me.

The memories of this period recovered in the analysis were as follows: When he went to Shreveport he had severe headache and felt worried and ill. After finding that he could do nothing to straighten out the oil deal, he wandered without definite aim to St. Charles, La., Beaumont, Galveston, Albion, Kingsville, and Brownsville, Tex., during the next three weeks, feeling despondent and suffering severely with headache. He then remembered that his wife did not know where he was, telegraphed her and went home. They then moved to Houston, where he felt too ill to work. He tried to obtain relief at the station of the Veterans' Bureau and wrote several letters to Washington, but was refused hospitalization. Then there was a period probably of about a week, the memories for which could not be revived. He next recalled a hotel in Dallas where he was under the care of a physician and registered by the name of John P. Ac. With him in his room was a woman who "seemed" to be taking care of him; he was unable to recall her name. After a few days he was better and managed to sell some blank oil leases which he had in his pocket, thereby securing about \$500. He did not worry about his past, he felt as if he had always been there, and accepted himself without question. At this time, he and the woman who had at first been taking care of him were living as man and wife. He was not sure, but he did not think they were married. He bought himself clothes and a suitcase and with Mrs. Ac., the name by which she went (the only other name he can recall is Honey), went to Amarilla; after a week he decided to go West. They went to Trinidad, Colo.; why is not clear, but probably because he heard of a job there. He secured a position as clerk and later stock-keeper in a wholesale grocery house, and he worked there for a year. He lived with Mrs. Ac. in a suburb four or five miles from the city, made a number of friends, and was here initiated into the secret order. He recalled that in applying for his position he stated that he was born in Chicago, that his parents were dead, and that he had been married before, his wife and two children being dead. As reference he gave his service in the navy and the work for the government during the Mexican trouble, although these memories of course belong to Ab. He was happy here, got along well

with Mrs. Ac., but drank a good deal. After a year he secured, through an advertisement, a better position in a grocery house at Casper, Wyo., where he worked for a year and a half and gave, as letters from the firm testify, excellent service. Then, not feeling well, he took a vacation with Mrs. Ac. and went to Trinidad, where they stayed for two weeks. He had been dabbling in oil leases, and on the day before he intended to return to Casper sold some for \$600. With this roll in his pocket he started for the station to buy transportation for the return at 5 the next morning. On the way he went into a saloon and bought a drink, flashing his roll of bills. Two men in the saloon left before he did and when he came out held him up, one of them striking him on the head with a gun and rendering him unconscious. The next memory is that of finding himself in the box car at Des Moines, N. M., with a growth of beard which suggested that he had been in the car for at least two days, ample time for the train journey between the two places.

With regard to the conditions which led up to this fugue, the memories for which were the last to return, it was learned that while in Louisiana, he discovered that his wife was corresponding with another man whom they had known since the days in Nashville. This worried him greatly, and, not knowing what to do, he wandered about in a dazed condition and finally moved with his family to Houston. There he saw a letter which was altogether "too familiar," quarreled with his wife and then disappeared. The truth of this story is established by the facts that after his disappearance the wife secured a divorce in the shortest time possible and married the man in question.

Examination.—Ab. presented no evidence of active disease, though the scars in the neck and thorax bore witness to the old tuberculosis. He was bright, alert, energetic and decidedly attractive. He was highly sensitive; tears were often observed in his eyes, emotional but not demonstrative. In spite of his roving life he appeared refined; he was courteous, and there was an entire absence of oaths or coarseness in his language. He was highly imaginative optimistic and easily became enthusiastic over plans for his future, especially in the oil business. He passed lightly over his failures and handicaps, and his judgment often appeared decidedly naive and childlike. During the earlier interviews, he frequently insisted on his lack of fear and his love for the adventurous, but when this was finally challenged and his tendency to run away emphasized, he admitted the truth of this contention, and then adduced occasions on which he had been very much frightened. It is also striking that he had had practically no quarrels or fights. The strong attachments of his life have been for his mother and deceased, eldest brother, both of whom have greatly aided and protected him. Outside the states of automatism, his relations with the opposite sex appear to have been decidedly conventional and not extremely ardent, but he was natural in his appetites and sex relationships. In the automatic states, especially when drinking, he was often a libertine.

During the analysis, he twice had recurrences of the "blinding headaches" which usually preceded the fugues. On one of these occasions, after recovering and refusing to communicate the memory of the existence of Mrs. Ac., he even started again on a fugue. He had gone to bed with a headache at 11:45, and at 12:15 found himself, fully dressed, in the kitchen on the way out of the house. He had a struggle before deciding to stay at home. It is also of interest to note that toward the end of the study he expressed himself as feeling, for the first time in many years, a "whole man"; and he wrote

spontaneously to Colorado to find out what had become of Mrs. Ac., to whom he realized he owed some adjustment, although at first his intention was to ignore this part of his life.

SUMMARY

The outstanding features of the personality of this patient are restless energy combined with a sensitive, timorous conventionality. Coupled with the associations of childhood, the former led to a craving for change and adventure which was held in check by the latter. The navy offered prospects of a successful compromise, which was shattered by illness with resultant intense resentment. At the same time alcohol was offered as a substitute and served for a time to allay the disappointment. With returning health this was discontinued, but a recurrence of the illness and its accompanying fears, serious psychic trauma, exhaustion and an accidental dose of whisky led to an effort to escape by a blind return to excessive drinking. Following this first fugue, the craving for excitement and escape from the trammels of his conventional inhibitions reappeared at intervals; and a feeling of illness offered a justification for resorting to alcohol, under the influence of which he gave way to sexual excesses, and finally married. To escape the consequences, he first drank until he developed delirium tremens, and then disappeared on a prolonged fugue. Subsequent difficulties have been reacted to in the same manner. The nature of these difficulties is sufficiently indicated in the foregoing detailed account and need not be further discussed here. It will be noted, however, that in many of the fugues there has been a concomitance with illness or injury as at the first attack. The reason for the recovery of the real personality is not always clear, but has usually been the presence of an illness or an accidental injury.

COMMENT

The mechanism underlying the condition seems thus to be quite clearly an escape from some situation intolerable to the sensitive personality, and thus brings the case under the category of psychoneuroses of hysterical type. The prolonged duration of the fugues and the ease with which the memories were recovered preclude any possibility of epilepsy.

DISCUSSION

DR. HUGH T. PATRICK: I think this is the first case of the kind reported to this society. The subject of fugues is a large one. There are various kinds occurring in several mental disorders, but in the type represented by Dr. Singer's case, I think it is clear that the patient is running away from something. He cannot adjust himself to the fight; he can neither dominate conditions nor endure them, so he runs away. The fact that he runs away in this state of secondary consciousness instead of consciously is due to his temperament or disposition plus his experience or training, just as some persons get out of difficulties by means of hysterical paralysis or hysterical fits. The reaction of

his particular temperament is to pass into a state of secondary consciousness and get away from trouble. Temperament and training probably also determine why these states of secondary consciousness last so long in some people and so short a time in others. Every one of ten or a dozen patients I have seen was escaping from something he did not like: financial difficulties, honest or dishonest, domestic troubles, irksome responsibility, punishment for petty crimes. One patient was, as nearly as I could find out, running away from a headache, and in Dr. Singer's case one of the first symptoms was headache. In some patients, alcohol seems to start the fugues. When sober they behave, but after a drinking bout they have a fugue. One case was that of a younger son of an English family who had traveled all over the continent while having fugues. He seldom went away for long periods unless he had been drinking. When I saw him he had come to himself, after a fashion, at a busy corner in Chicago. He stopped a policeman and asked him where he was, and the policeman named the corner. He then said, "What city is this?" When told he next asked what his (the patient's) name was. The policeman was inclined to be irritated, but then saw that there was something wrong with the man and took him to a hospital, and it was a long time before he could remember his name. After he had been in the hospital for some time he told us a name. It proved not to be his name. After a week or so he said, "Now I know my name," and gave a totally different name, but nothing could be found to connect him with this name. After a few days he still insisted that that was his name and recalled a Chicago man with whom he had gone to college. I called up this man, and he said he had never known such a man at college. I took the message back to the patient who was puzzled and indignant and said, "Of course he knows me; now you go tell him about the time he and I and A—— B——, did so and so," relating in detail a college escapade. The Chicagoan remembered the incident very well but said the third man was X—— Y——. On receiving the message the patient said, "Of course, *that's* my name!" and we then traced his relationship and found out who he was. That fugue started in an intoxicated state. He had been attending the international yacht races, had been drinking a lot of champagne, and the next thing he knew he found himself in Chicago. He always had something to run away from; he was always in trouble from indulging in more or less irresponsible irregularities. I have never had a case with such long absences as the one reported by Dr. Singer, although there are many in the literature.

Some years ago I had occasion to look up the literature rather fully, and the conclusion that I came to, and which I feel certain is right, is that an epileptic fugue is always exceedingly brief. I do not believe that ambulatory automatism due to epilepsy ever lasts more than a few hours, certainly not more than one day; that is, a fugue in which the patient acts in a natural and normal way. One of the very early cases is one reported by Charcot, but after a study of his report and the reports of others subsequently on the same case, it seemed clear that it was not an epileptic case at all. I was unable to find in the literature a single conclusive case of epileptic ambulatory automatism, or what seemed to me to be that, that lasted more than a few hours. I have never had one in my own experience. I should be very much pleased to have Dr. Singer's opinion on this point.

DR. PETER BASSOE: I was struck by the history of headaches in this case which recalls the possible relationship between the three conditions: migraine, alcoholism and epilepsy. We all know that Kraepelin looked on dipsomania as

a form of epilepsy, and I would like to ask Dr. Singer whether it might be assumed that these fugues were a sort of epileptic equivalent. Then the only peculiar thing would be their long duration.

DR. HERMAN M. ADLER: I think one thing should be emphasized: I believe that we all agree with Dr. Patrick that this case is one of an attempt to escape, but it is important to note that it is an attempt to escape from something in the person himself and not from something outside of himself. Dr. A. W. Stearns has recently shown in his work on suicide that suicide may be interpreted as an attempt to escape from the ego. It is interesting to note that in Dr. Singer's case we are dealing with an intelligent and very sensitive person. This is according to the general rule, that the more intelligent and sensitive the patient, the more likely he is to find intolerable situations. One would expect to find such conditions rather frequently among criminals, yet that does not seem to be the case. Not infrequently some of our prisoners present a good imitation of fugue, but we are able to analyze the case and find that it is one of shamming rather than a true fugue. Of course, there are a few cases in which a true fugue occurs. Criminals, as a rule, are not sensitive, even though they may be intelligent.

DR. H. DOUGLAS SINGER: The question of epilepsy has been fairly well covered. I may add that the memories of a period of epileptic automatism are extremely difficult, if not impossible, to recover. In this man such recovery was easy. He readily passed into a hypnoidal state, though many memories were revived during full waking consciousness.

The view expressed by Dr. Patrick is well illustrated by a case seen recently in the Cook County Hospital. A boy was brought in by the police who did not know a thing about himself. His past was an absolute blank. I saw him the day he was brought in, and after a few simple questions on school knowledge happened to ask the right question, "What are you trying to forget?" He stared at me, tears appeared in his eyes and he said, "Now I remember! My girl has just turned me down." He still maintained that he did not know his name or past, and I gave him five minutes to think. When I returned he had recovered full memory. About a year before he had been gambling, had borrowed money, was put on probation in the Boy's Court and ordered to repay in weekly installments. He succeeded until he fell in love and wanted to spend money on the girl. She, in some way, learned the story and "turned him down." In his fugue the same day, he was trying to forget the whole situation.

I can see no advantage in attempting to connect such episodes with epilepsy. In the case here reported the fugues, including the headaches and alcoholism, seem to be clearly efforts to escape from intolerable situations by ignoring them, a mode of reaction to which some types of personality are more liable than others, particularly, as Dr. Adler says, the sensitive timidity so well illustrated by this patient.

BASAL METABOLISM IN MENTAL DISEASE *

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BOSTON

Heretofore variations in basal metabolism have been considered essentially as an index of the activity of the thyroid gland. We have correlated a series of fifty cases in which tabulations were made and which for the most part showed no clinical evidence of thyroid disease.

Great stress has been laid in the past on the finding of an increased metabolic rate associated with hyperthyroidism. In the present group of cases, which are unselected, a striking feature is the predominance of low metabolic rates. The significance of this rate is undetermined, and we present these data without venturing to interpret them.

The apparatus used in these investigations is the so-called "Benedict portable" manufactured by W. E. Collins. This machine measures only the oxygen consumption. The sources of error in the use of this machine in our patients are: 1. Soda lime must be replaced frequently. 2. Leaks in the system must be looked for in every determination (in dealing with mental patients one must especially look for leaks around the mouth piece). 3. The cooperation of the patient must be carefully determined in regard to both activity and forced breathing.

In every case at least two determinations checking within 5 per cent. were used. The Du Bois formula was used in estimating the body surface.

Little has been written on the subject of basal metabolism in mental disease. In a recent article,¹ one of us has reported low basal metabolism in ten cases of dementia praecox. Kirby and Gibbs² likewise report a series of ten cases of dementia praecox and a number of manic-depressive cases of various types. In a large number of their cases readings were normal, but there was a definite tendency toward low readings. It is interesting that the only case in their series that gave a markedly high reading was a case of mixed type of manic-depressive psychosis.

In our series of fifty cases, only four showed increased basal metabolism, if we accept ± 10 per cent. as the normal variation. Three of these cases belonged to the manic-depressive group, two were cases

* From the Laboratory of Internal Medicine, Boston Psychopathic Hospital.

1. Bowman, K. M.; Eidson, J. P., and Burladge, S. P.: *Biochemical Studies in Ten Cases of Dementia Praecox*, Boston M. & S. J. **187**:10, 358 (Sept. 7) 1922.

2. Kirby, G. H., and Gibbs, C. E.: To appear in the *Archives of Internal Medicine*.

BASAL METABOLISM IN VARIOUS MENTAL DISEASES

Case No.	Pulse	Nutrition	Age	Sex	Basal Metabolism	Diagnosis
1415	74	W. N.	43	F	-25	Traumatic psychosis
2064	80	W. N.	21	F	-3	Psychosis with cerebral syphilis
2168	74	W. N.	47	M	-15	Psychosis with brain tumor
796	86	F. W. N.	49	F	-11	Psychosis with lethargic encephalitis
3533	72	W. N.	27	F	-21	Psychosis with lethargic encephalitis
3511	72	W. N.	29	F	-21	General paresis
		W. N.	67	M	-17	Psychosis with organic brain disease (type undetermined)
1768	100	F. W. N.	23	F	-17	Psychosis with organic brain disease (type undetermined)
1574	104	W. N.	14	F	-17	Postoperative delirium, partial thyroidectomy
1789	80	F. W. N.	30	F	-15	Manic-depressive mania
2856	120	W. N.	19	F	+2	Manic-depressive mania
1663	96	F. W. N.	21	M	+13; +2	Manic-depressive depression
3352	120	W. N.	19	F	-2; -10	Manic-depressive depression
1693	76	W. N.	19	M	+15	Manic-depressive depression
2475	76	W. N.	33	F	+77; +66; +40	Manic-depressive mixed
1286	94	P. N.	15	M	-21; -31; -37	Schizophrenia
2068	120	P. N.	14	M	-11	Schizophrenia
3370	78	W. N.	16	F	-20; -17	Schizophrenia
3464	84	W. N.	15	F	-14	Schizophrenia
3100	74	P. N.	24	M	-3	Schizophrenia
2281	80	W. N.	35	F	-5	Psychosis with mental deficiency
1874	100	W. N.	17	F	-8	Undiagnosed psychosis
3395	70	W. N.	40	F	-26; -27	Psychoneurosis; hysteria
3480	92	W. N.	21	F	-25	Psychoneurosis; hysteria
3469	82	W. N.	20	F	-6	Psychoneurosis; hysteria
3376	80	F. W. N.	28	M	-18	Psychoneurosis; neurasthenia
1549	84	W. N.	25	F	100	Psychoneurosis; anxiety type
2308	58	W. N.	40	M	-16	Psychoneurosis; anxiety type
1766	80	W. N.	26	F	-1	Psychoneurosis; anxiety type
1845	90	W. N.	20	F	-5	Psychoneurosis
2910	70	W. N.	21	F	-12; -18	Narcolepsy
		W. N.	12	F	-20	Epilepsy without psychosis
2417	68	W. N.	8	M	100	Epilepsy without psychosis
1878	74	W. N.	10	F	-21	Epilepsy without psychosis
3084	76	W. N.	19	M	-7	Epilepsy without psychosis
2177	72	W. N.	12	M	-20	Epilepsy without psychosis
2766	84	W. N.	21	F	+8	Epilepsy without psychosis
2328	76	F. W. N.	14	M	-13; -2	Psychopathic personality without psychosis
1933	80	W. N.	25	F	-6	Psychopathic personality without psychosis
2193	80	W. N.	19	M	-8	Psychopathic personality without psychosis
1455	84	W. N.	25	F	-14	Psychopathic personality without psychosis
964	74	W. N.	18	F	+15; +2	Psychopathic personality without psychosis
3104	90	W. N.	18	F	-12	Psychopathic personality without psychosis
3580	68	F. W. N.	23	F	-16	Psychopathic personality without psychosis
3266	88	W. N.	15	F	-5	Mental deficiency without psychosis
3069	96	W. N.	58	F	-6; -6	Mental deficiency and cretinism without psychosis
3072	80	W. N.	11	F	-6	Mental deficiency without psychosis
G 4	...	W. N.	50	F	-18	Myxedema
G 2	...	W. N.	45	F	-33	Myxedema
Q 5	...	W. N.	50	F	-17	Myxedema

of depression with readings of + 13 per cent. and + 2 per cent. in one case and + 15 per cent. in the other; one, a mixed type, had readings of + 77 per cent., + 66 per cent. and + 40 per cent. It is, perhaps, noteworthy that the only case in the entire series with a markedly increased basal metabolism was this case of the mixed type of manic-depressive psychosis, since Kirby and Gibbs found the same to be true in their series. The fourth case with a high reading was one of psychopathic personality without psychosis, in which the basal metabolism was + 15 per cent. and + 2 per cent. Two more cases had plus readings, but within normal limits, and two cases gave readings of exactly 100 per cent. The remaining forty-two cases all gave minus readings and twenty-seven of these, over half of the cases in the entire series, had a basal metabolism of less than — 10 per cent., which is commonly considered the lower limit of normal.

It seems worth while to call attention to the fact that eight out of nine patients with organic psychoses had abnormally low basal metabolism, that the tendency toward low basal metabolism previously reported in schizophrenia is confirmed by the few cases of this series, and that the manic-depressive group showed less tendency toward low readings than any other group.

We cannot but feel that this tendency toward a low basal metabolism in cases of mental disease is of importance and merits consideration in formulating our theories as to etiology and treatment. On the other hand, we do not feel that any conclusions are justifiable as yet. Careful physical examination and administration of thyroid extract by mouth or thyroxin intravenously have made us feel that simple hypothyroidism is not the explanation of the large number of cases of abnormally low basal metabolism. Kirby and Gibbs have reached the same conclusion. Further work along this line is being done, and we hope ultimately to settle this point. On the other hand, Lewis and Davis³ report that they found evidence of hypothyroidism in sixteen out of twenty-two cases of schizophrenia of from one to ten years' duration, and that following thyroid feeding, nineteen patients improved physically, four recovered mentally and were discharged, nine improved mentally and nine did not improve mentally.

Except as noted in the list of diagnoses, in no case were there physical findings which made us suspect endocrine disturbance.

Since inanition has some effect in lowering basal metabolism, we have recorded the degree of nutrition in each case in the table. *W. N.*

3. Lewis, N. D. C., and Davis, G. R.: A Correlative Study of Endocrine Imbalance and Mental Disease, *J. Nerv. & Ment. Dis.* **54**: No. 5; **54**: No. 6, and **55**: No. 1, 1921, 1922.

means well nourished; *F. W. N.*, fairly well nourished; *P. N.*, poorly nourished. It will be seen from a study of the table that only three cases showed sufficient inanition to affect the basal metabolism.

We have also recorded the pulse rate, age and sex in each case, feeling that they are of sufficient importance to warrant so doing.

Abstracts from Current Literature

THE ORIGIN OF PSYCHIC ENERGY. J. DANYSZ, Paris: J. B. Baillière et Fils, 1921. Abstract translation of pages 113-118.

It is well worth while for us who trudge or speed along special roads in the domain of biology, occasionally to take a journey across country into broad fields and across new roads. Many books are now written to guide us on such journeys, and of these Dr. Danysz' "Genesis of Psychic Energy" is of especial interest to neurologists. We probably will not agree in detail with his view of this genesis, but at least it is broadening and brings to mind possible neurologic bearings of recent advances in physics, chemistry, genetics, serology and anaphylaxis. For example, few of us will agree with his statement that "In an animal, a change caused by a factor from the exterior can only become durable and transmittable if it causes a new equilibrium in the functions of the endocrine glands." This may be the basis of creative evolution, but as yet there is not enough evidence to warrant such a statement. Nevertheless, the book contains enough stimulation to make it of definite value. A summarized translation follows:

Our conception of the constitution and evolution of the universe rests on the analysis and coordination of the following facts:

The universe is composed of a certain number of units: matter, distinct energy, which can be divided into two classes:

1. Units which seemed indivisible, whose duration used to be considered unlimited and whose structures appeared constant and invariable, namely, the atoms: hydrogen, oxygen, nitrogen, carbon, iron, etc.

2. Units which undergo incessant transformation and are influenced by the variation of their surroundings.

The discovery of radioactive bodies and the spectroscopic study of the stars has further subdivided atoms into electrons, with provision for a further subdivision into etherons, so that we can place the electron or the etheron at the base of progressive evolution as the unit of primordial matter-energy.

In the "necessary succession" in the formation of "matter-energy" units of greater and greater power by the reunion and coordination of more simple units into more complex ones in the inorganic world and by the multiplication and differentiation of cells in the living world, we consider the following factors to have been potent:

1. The gradual and continual lowering of the temperature in the inorganic period. This factor contributed toward selection of the less stable and more active elements which could only thus become the components of more complex units.

2. The formation of water into a liquid state on the surface of the earth and the progressive lowering of the temperature of this water to about 50 degrees was the factor in the second selection. The water acted as a solvent of different elements and compositions, in different proportions, and as a catalyser of dissolved and dissociated elements in the proportion in which they were necessary to form living matter. The formation of the first living units did not require the intervention of a special energy. It was the natural consequence of a series of selections which ended in the reunion

of the lightest elements and those richest in multiple affinities and capable of forming the less stable compositions. These, by the sole play of their affinities, aided by the catalysing properties of the water, were able to give birth to the first living micelles.

3. Once living beings are constituted, with an interior milieu of infinite complexity and with a great sensibility to all the actions of the exterior milieu, it is the radiating energies which become the most active factors in progressive evolution. They cause the formation of sense organs and, later, of nerve tissue.

4. This nerve tissue and its different forms of energy have brought this evolution up to man, that is, to a brain, in which the spheres of association have acquired a development dominating all the other parts of the encephalon. From the brain is derived a specific energy—psychic energy, which results from the coordinate cooperation of all the preceding “matter-energy” units, and which has become so powerful that it does not passively submit to the reactions of the exterior milieu, but which, having become conscious of itself, of its force and means of action, tries to intervene actively in the evolution of its interior milieu and to adapt to it the exterior milieu.

This evolution is progressive because, at each degree of complexity, the energetic power of the newly formed units augmented in ever increasing proportions, and this increase of power was a necessary consequence of the succession of the coordinated associations of all the preceding “matter-energy” units, which do not simply progress numerically, but organize for common action.

After the appearance of mammals, one of the most important factors in the continuity and rapidity of evolution was the constitution of an increasingly constant temperature in the interior milieu, an indispensable condition for a regular continuity of reactions and of a strong resistance to the disturbing influences of temperature variations in the exterior milieu.

From the preceding facts we can conclude that living matter, considered as a chemical composition, conforms to the general laws which determine the reactions of mineral matter composed of the same simple elements; considered as a physiologic unit, it obeys laws which are not exclusively within the domain of pure chemistry, mineral or molecular.

Each new unit formed by inorganic synthesis or by a biologic differentiation and endowed with a new form of energy has a right to a new name to characterize its structure as well as its function. In micelles chemical affinity does not obey the same laws as in molecules, and it would be foolish to call units endowed with different properties by the same name. Excepting colloids, which gave birth to the first albuminous micelles, and through them to living cells that we will call the first biologic units, we can assume that the first important factor in the progressive evolution of living matter was the formation of vitamins by protozoas and by vegetables. We have observed, as much as it is possible to judge by present experiments, that it is entirely due to vitamins that animals, which are incapable of producing them themselves, can assimilate the vegetable albumins and nourish themselves thus with substances already selected (and of a more elevated structure) and acquire from them a greater activity and plasticity, which permit them to submit to the influence of radiating energies and finally to differentiate nerve tissue.

The second determining factor was the formation, by differentiation, of “nerve tissue” first, of the peripheral organs of sense, then of nerve fibers, then of ganglions or nerve centers and finally, in these centers, of sensory

spheres and more and more complex and extended spheres of association. In proportion as organisms became more and more complex, because more finely differentiated, they became at the same time more and more sensitive to increasingly numerous and varied substances and forms of energy. Thus, nearly all living beings—animals, protozoas or vegetables—are nearly equally sensitive to simple crystalloids (acids, bases, salts); complex crystalloids (glucosids, alkaloids) are of a more selective action; they act only in a much larger dosage on vegetables and the lower animals than on the higher forms of animal life. Colloids probably provoke no appreciable reaction on saprophytic vegetables and react only slightly on unicellular beings, vegetable parasites and the lower animals. The formation of antibodies appears to cause important changes only in the higher vertebrates. Radiating energies only become dominant factors in evolution among animals with a differentiated nervous system, and their action is more pronounced (particularly that of light and sound) in proportion as the peripheral organ and the corresponding sensory center are more developed. The emotions do not begin to be felt and to react except on animals provided with a brain, and finally so-called thought, conscious will and its ends, appears and begins to react in an appreciable way on evolution only with the formation of the spheres of association in man. The appearance of thought marks the most important epoch in the process of progressive evolution, because this form of energy reacts not only on the evolution of the "matter-energy" unit man, on his interior milieu, but also, in an indirect way, on his entire exterior milieu, on all the "matter energy" units which have preceded man.

Of course, all these actions and reactions have, as a starting point, physico-chemical reactions and end in other reactions of the same nature; it would be of the greatest interest to know their intimate mechanism. We know what goes into an organism and we can analyze what comes out, but we are in complete ignorance of what transformations each element undergoes and forms during its passage, because we do not know the intimate constitution of the physiologic unit of every living being, Darwin's gemmule, Elseberg's plastidule, de Vries' pangènes, Wiesner's plasome, Hertwig's idioblaste, Weissmann's biophore or déterminant, etc., in short, of the living albuminous micelle, which cannot, by its definition, have the same chemical composition nor the same structure as the dead albuminous micelle. Clarke has made wings grow on a wingless bug by watering the rose bushes with a little magnesium sulphate, and Stockard raised fish with one eye in the middle of their heads by adding a little chlorid of magnesium to the water, but we do not know the cause nor the mechanism.

It is all the more difficult to determine the cause of these extraordinary phenomena because other substances of a very different nature chemically can present the same effect. The only conclusions we can draw are that the effect probably results from a series of indirect reactions, the hyposecretion or hypersecretion of a gland which determines the formation of certain organs or tissues, or a combination of the foreign substances with the hormone or hormonozone already in the blood or humors. The effect produced will be temporary or permanent, the new character will be individual or hereditary according to whether the reaction provoked is more or less durable and whether it has exercised a corresponding change in the cells of reproduction. Organic colloids, albumins, by the formation of antibodies, cause the most durable changes in the organism: if the new equilibrium is favorable to the development of the individual, the race becomes powerful; if the contrary, the race

may disappear. The equilibrium among the various organs in an organism must be as perfect as possible in order that the individual and race may be strong.

Among the higher forms of animal life, every evolutionary reaction, ascending or descending, is effected either by the action of the nervous system, or (and this is the most frequent case) by the intermediation of the glands of internal secretion which regulate the conditions of nutrition and through that the development of all the other tissues and organs. In an animal, a change caused by a factor from the exterior can only become durable and transmittable if it causes a new equilibrium in the functions of the endocrine glands.

Summary.—All that we know today about the progressive evolution of living matter can be summed up in a few words:

Since the formation of the first biologic units of unicellular beings, living matter continues today to develop and to grow by the multiplication of unicellular organisms. Its continuous progressive evolution consists essentially in the progressive formation for this unicellular organism of an immediate milieu of evolution, increasingly differentiated and endowed, thanks to this increasing complexity, with an energy of its own, more and more powerful and becoming therefore at the same time more resistant, more stable and of a more delicate sensibility to the action of the ever variable elements in the exterior milieu. This evolution has been attained by the alternative formation and coordination of structures and of new forms of energy, step by step, in an uninterrupted fashion, through millions of centuries, in a spontaneous and unconscious way, up to man, who becomes more and more conscious of the mechanism of this evolution, of the biologic rôle of the imperishable unicellular organism and of its immediate milieu, the perishable individual.

And, thanks to this new energy, consciousness, resulting from the coordination of structures and from more and more differentiated energies, man seeks to intervene in the knowledge of cause and effects and in the future mechanism of this progressive evolution, to dominate and to conduct it with more security and certainty in the direction that it seems to be taking by itself. This is the direction that the knowledge of the past indicates as the only possible one: the unlimited growth of psychic energy and the unlimited differentiation of its organ, the brain.

Thus man has arrived at the conception of a definite aim for his efforts, an ideal for the future realizable from an unlimited evolution of the body and the spirit, because this evolution carries with it all the elements of an infinitely perfectable structural and energetic growth.

SUSAN E. HALE and STANLEY COBB, Boston.

DYSTONIA LENTICULARIS PROGRESSIVA. ARRIGO FRIGERIO, *Riv. di patol. nerv.* 27:1 (Oct. 15) 1922.

The author reports a clinical case in detail. The patient, aged 51, a Jew, who was quite alcoholic, began to show mental peculiarities in 1902. He had trouble in beginning speech, and syllable stuttering with slowness of speech was noted. There were vomiting, polyuria and polydipsia. In 1908, he complained of headaches, and one night he was found comatose on the doorsteps of his home. He was stuporous for twenty-four hours, and then he had an epileptiform convulsion lasting one-half hour. He recovered completely in a short time. The urine contained sugar at this time, and he was treated for diabetes, but this diagnosis was changed, and the patient was placed on

a full diet. Some time later he walked badly, stumbled and complained also of fornication and pains in his legs, especially in the left knee. At this time the patient also noted fibrillary contractions in the muscles of the lower left extremity, and a gradually increasing atrophy. In February, 1921, his complaints were of the motor disturbances described later, and above all, severe pains in the left knee.

Examination revealed normal intelligence. Speech was slow with syllable stuttering. The patient walked with his right hand strongly grasping a cane. He inclined forward, trying to steady himself with his left hand, and when he descended stairs, the left leg was often violently thrust forward, remaining momentarily in the position of strong extension at the knee and flexion at the hip. At other times, the hip would be suddenly extended and the whole foot slapped to the ground. Occasionally the leg would become briskly flexed in all its joints, giving a typical steppage gait. When the patient was seated, inconstant brisk movements of flexion and extension of moderate excursion of the leg on the thigh and the feet on the leg were noticed.

Similar movements of adduction and abduction or of internal and external rotation of the various segments of the lower extremities were noticed, especially in the feet. Occasionally a moderate extension of the foot on the leg and of the leg on the thigh with flexion of the latter on the pelvis was noticed, this movement being bilateral and strong enough to hold the extremities at right angles to the pelvis; the position was held briefly, subsequent relaxation allowing the extremities to return to a normal position. Small movements of flexion and extension of the fingers, especially of the terminal phalanges, were noticed, particularly in the left hand; these movements were sometimes slow, and sometimes brisk. Sometimes small fibrillary, clonic movements or fascicular twitchings of irregular frequency and rhythm were noted, especially in the hypothenar eminence. Similar movements were noted in the calves and in the toes, especially the left. The movements of flexion and extension, adduction and abduction and rotation with the attitude of equinovarus were marked. Particularly strong irregular movements were noted in the bellies of the quadriceps, which would quickly be relaxed. This phenomenon was less marked in the right thigh. Occasional movements of similar character were noticed in the abdominal muscles, and also brisk contractions of the cremasterics or of the glutei and other pelvic muscles. Sometimes these constant involuntary movements began in waves with small localized twitchings in the left quadriceps, then larger stronger contractions of longer duration occurred. Finally the myoclonic jumps and waves of fascicular contractions or even fairly prolonged contraction of the entire quadriceps of the right side appeared; then brief, brisk contractions of one or the other thigh, and later a movement of relaxation and rest, with the quick resumption of the fibrillary and fascicular clonic movements. Occasionally there were interposed contractions of the glutei, rotators, and flexors of the pelvis, with strange contortions of the pelvis.

Neurologic examination also revealed the sluggish reactions of both pupils to light, the left pupil being larger. There was some weakness in the left hand, as shown by the dynamometer (the right hand registering 42 and the left 35). There were slight dysmetria and asynergia in the left upper extremity, and marked atrophy in the left lower extremity (2 cm). There was hammer toe deformity of all toes of the left foot. There was some dysmetria in the left lower extremity, although it was difficult to examine this because of the rarely occurring quiet moment. The patient could not elevate either extended

lower extremity alone without immediately causing brisk movements of abduction and adduction, but both limbs together could be held elevated in extension. The patient was better on some days than others. Emotions and many stimuli made him worse. The involuntary movements disappeared in sleep but began a few minutes after he began to walk in the morning. There were no disturbances of sensation except a slight uncertainty about describing the position passively assumed by the various segments of the lower extremities. The roentgen ray showed a small liver shadow.

The laboratory examinations showed a negative Wassermann reaction in the blood and spinal fluid. The fluid was under normal pressure, and the Nonne-Apelt reaction was negative; there were three or four cells in each field (Ravaut).

Later the patient developed a marked lordosis and a marked steppage gait. Finally, bizarre attitudes of the entire trunk and extremities were assumed when walking. The speech became progressively more explosive and staccato. There seemed to be a brisk contraction of the muscles in inspiration thus causing the speech difficulty. The deep reflexes were always almost impossible to obtain, although it was definitely determined that they were present during his fleeting moments of rest. Those of the right side were slightly more active but the Achilles reflex was never obtainable. The author states that the patient had no paralysis following the onset with stupor in 1918.

Disregarding the fact that there is no evidence whatever that the patient fell or had any trauma, the author feels that the cause of his patient's trouble was precipitated by trauma acting in an old degenerated (alcoholic) abiotrophic nervous system. The tonic and clonic movements are explained by spinal, cortical or subcortical localization, while the disturbances of posture, atasia and the dysmetria incriminate the cerebellar system; the atrophy in the left leg is explained either by peripheral or central involvement. The author eliminates the possibility of a neuritis, and because it was impossible to obtain electrical reactions, he leaves the latter question unanswered.

In making a differential diagnosis, the author eliminates tabes, multiple sclerosis, the syndrome of Benedict, the paramyoclonus of Friedreich and Huntington's chorea. He considered chronic chorea and myoclonus epilepsy, and after comparing his case with the reported cases of dystonia muscularis deformans and Wilson's disease, he excluded both of these, and finally concluded that it was a case of dystonia lenticularis progressiva. The author feels that his case has many points of contact with Gowers' case as quoted by Wilson.

Considerable importance is attached to the fact that the patient showed traces of glycosuria (4 per cent.) for many days after the administration of 50 gm. of saccharose by mouth. This did not disappear until a carbohydrate restricted diet was given. In this connection is quoted the work of Roel and Lewy, who showed histologic alterations in the globus pallidus in four cases of diabetes. The fact that the roentgen ray showed a small liver shadow in the author's case is cited as an argument for placing the lesion in the lenticular nucleus.

While the author agrees that one should not attempt, as many have done, to place Wilson's disease, chorea, athetosis, paralysis agitans, Little's syndrome, tuberous sclerosis, etc., in the striatum, he feels that because of Hunt's work, his case probably belongs in the striatal group. He agrees with Hunt that the tremor, rigidity and dysarthria are part of a globus pallidus syndrome, while the putamen may have ascribed to it the involuntary tonic and clonic

contractions and the athetoses. This conception explains Wilson's disease, wherein there is degeneration of the entire lenticular nucleus. He recalls that Mingazzini localized the pathologic condition in cases showing monotremor of the forearm and hand in the medial portion of the putamen of the opposite side, the dysarthria being due to lesions of the putamen of the left side. In these cases, there is atrophy in the paretic extremities. In his case, the author feels that all of the symptoms, including the atrophy of the left leg and the epileptiform convulsions, can thus be explained. He finds difficulty in explaining the dysmetria and asynergia, except on the basis of the connections of the corpus striatum with the cerebellum, which he thinks are involved in his case.

OSNATO, New York.

SOME ENDOCRINE ASPECTS OF THE PSYCHE. (A LECTURE DELIVERED TO THE UPSALA MEDICAL SOCIETY.) J. A. HAMMAR, *Folia neuro-biolog.* **12**:2, 1922.

Localization of different psychic processes depends to a great extent upon the structure and function of the nervous system. The author confines his article to the functional aspect of the question, and emphasizes the fact that the psychic processes are to some extent dependent on the reaction of the nerve elements to chemical influences.

Attention is called to the psychic alterations produced by the implantation of glands of the opposite sex in experimentally castrated animals. Steinach's case of the homosexual man in whom the testicle from a psychosexually normal person was implanted with successful results, is cited. The psychic changes occurring at puberty and the menopause, during pregnancy, and following castration, emphasize the rôle of the sex glands. The effects of thyroid feeding in cretinism, the mental reactions in exophthalmic goiter, sometimes a psychosis, and the syndromes found in acromegaly, Addison's disease and diabetes, furnish examples of the important influence which internal secretions have on nervous functions. Farrant's reports are reviewed, and the opinion is quoted that toxemia is the primary factor which influences mentality through the endocrine organs.

An elective hormonal action must be imagined which affects the elements of the nervous system in different ways in accordance with the varying type of hormonal secretion. It is probably a question of chemical affinity and chemical stimuli, of an electivity in the relations of nervous elements to chemical substances. The action on the nervous system of epinephrin, curarin, physostigmin, hypnotics and other substances, illustrates this electivity from the standpoint of pharmacology. Strychnin has an affinity for the motor parts of the brain; tuberculin, mallein, and diphtheria toxin become especially fixed to the lipoids, and tetanus toxin has an elective affinity for the albuminoids.

The activity of the endocrine substances in the system is directly analogous, but they are endogenous instead of exogenous. The cell is considered to be the point of election though the synapsis cannot be definitely ruled out. Sleep, as explained by Pitrooy, depends on a "hypnotoxin" which is probably formed in the brain. Mention is also made of the theories about the function of glia cells, as to whether this is merely a supporting tissue or has a nutritive or trophic function.

The author considers that neurologic inferiority, either acquired or inherited, must rest on a chemical foundation. There is a wide range of individual susceptibility to certain poisonous substances such as alcohol, nicotin, caffein,

etc., an idiosyncrasy which may be biochemical as well as biophysical in basis. The internal secretions, as chemical agents, markedly alter various somatic conditions, and produce changes in external appearance, as well as in psychic activity. Just how this takes place is, at present, mere speculation, because little is known of the functional interrelationship of the various glands.

The rôle of endocrine substances in emotional life has been investigated and it has been shown, for instance, that the thyroid and adrenals are largely concerned in the syndromes of anger and fear. Here the nervous system must be responsible for the glandular activity, but the manner of the innervation of the glands is based upon indirect observation. There has not been shown a double innervation—stimulatory and inhibitory—as in the case of the exocrine organs.

The central nervous system as the seat of origin of emotion is cited in contrast, and the statement is made that "the central excitation may be the primary factor and the endocrine mechanism may be stimulated through the autonomic nervous system." Cannon's experiments on the adrenals and thyroid, which test the reactivity through nervous stimulation, and the effect of the gland substances in the blood stream, are taken as authoritative.

Visceral alterations, either disease or malfunction, have a direct influence on psychic reactions and have been observed by every physician. But in them, there is the sensation produced by the organ disturbance, in addition to the psychic manifestations. In the endocrine organs no sensations of the structures themselves are ever manifest and the reaction is mainly psychic.

PATTEN, Philadelphia.

MENTAL HYGIENE NEEDS ARISING SUBSEQUENT TO SCHOOL LIFE. C. FLOYD HAVILAND. *Ment. Hyg.* 6:688 (Oct.) 1922.

This author gives first place in the mental-hygiene program to the period of childhood as the golden period for preventive work; yet he points out that until such preventive work shall have become far more effective than it is at present, the mental-hygiene needs of post-school life will constitute a vast social problem that must be met, for the sake of the community as well as for that of the persons concerned.

The extent of the problem is such that the state must necessarily play the chief rôle in dealing with it; and its most obvious duty in this respect is the provision of adequate facilities for the treatment of those mental patients who are legally termed insane. No state has yet been able to make such provision, owing to the increase in the number of known insane—an increase probably representing in part an actual increase in insanity, possibly due to economic depression, and in part an increased public confidence in state institutions. The net increase of patients on the books of the New York state hospitals for the fiscal year ending June 30, 1921 was 1,442, the greatest increase of any year in the history of the state hospital system. New York is endeavoring to meet this condition by a construction program that allows for a certain additional number of beds every year, and by a liberal use of the parole system; the number of patients on parole from the state's civil hospitals has increased by nearly 1,700 in the last five years. Besides the provision of a sufficient number of beds, the state should render the services of the state hospital readily available through laws permitting emergency commitment and voluntary admission under proper conditions.

While the state hospital should be essentially a curative institution, its functions include the partial rehabilitation of so-called chronic mental patients through

habit training and occupational therapy. This applies especially to patients suffering from dementia praecox, who may often be transformed from total liabilities to at least partial economic assets.

The extramural activities of the state hospital are no less important than the intramural. They include the placement and supervision of discharged and paroled patients, the treatment of incipient cases of mental disease in the community, and the dissemination of information on all matters pertaining to the maintenance of mental health. The outpatient mental clinic and its associated psychiatric social-work department are indispensable for this extramural work.

While the mental hospital should be prepared to treat all types of mental disease, there is a large field of usefulness in the immediate treatment of acute mental cases for the psychopathic hospital, conducted on the same basis as the general hospital, and for the psychopathic ward in the general hospital. This placing of mental disease on the same footing as other diseases will do more than anything else to change the old fatalistic attitude toward mental trouble.

In conclusion, the author emphasizes the fact that no public health movement is complete without a mental hygiene program; a human being is a psychobiologic unit in which psychic and physical elements are interdependent, and health implies harmonious adjustment, both physical and mental.

WILLIAMS, New York.

TWENTY CASES OF LETHARGIC ENCEPHALITIS WITH PATHOLOGICAL FINDINGS IN FOUR CASES. W. E. HUME, F. J. NATTRASS and A. F. B. SHAW, *Quart. J. Med.* **15**:131 (Jan.) 1922.

This is a summary of twenty cases of lethargic encephalitis seen by the authors. There were fourteen males and six females; the ages varied from 6 to 60 years; the occupations were chiefly of artisan kind, one was a farmer; seventeen were urban residents and three rural; only one patient was known to have been in contact with other cases. In twelve cases the onset was acute, and in eight insidious. In five acute cases the initial rise of temperature was quickly followed by delirium. Three cases suffered with fever and neuritic pains. In the insidious cases the onset occurred with a gradually increasing drowsiness which continued for a few days to five weeks. Twelve patients suffered with diplopia, and six of these had a definite ocular palsy. In two cases there was bilateral ptosis. The pupillary reactions varied; three cases showed complete inactivity to light and accommodation; sluggish reactions were noted in five cases; in five the pupils were unequal but reacted to light and accommodation. In no case was there any evidence of optic neuritis even on repeated examinations. Nystagmus was present in two cases. Weakness of the muscles supplied by one seventh cranial nerve was noted in five cases. The majority of the cases showed restricted movements of the facial muscles ("Parkinsonian mask"). The right side of the tongue was paralyzed in one case. Fine tremors of muscle groups were present in four cases. Myoclonic movements were noted in fourteen cases (limbs nine, abdomen and back three, face and jaw two). Rigidity was present in five cases. The patella jerks were not obtained in one case; they were sluggish in one case, unequal in three cases, and exaggerated in one case. Three cases showed bilateral and three a unilateral Babinski sign. In one case there was complete retention of urine, in one case occasional retention and in three

cases temporary incontinence. The cerebrospinal fluid was examined in eight cases: one developed a coagulum; pleocytosis was present in four cases, an excess of sugar in two cases, and an excess of globulin in two cases. All Wassermann reactions were negative. Four deaths occurred on the fifteenth, twentieth, twenty-second, and thirtieth day of the disease. One case recovered in a week, one in six weeks, six in six to twelve weeks, and seven were convalescing for several months. Details are given of the history and symptoms of the four cases which resulted fatally together with a brief summary of the gross pathologic and histologic findings. None of these cases show anything hitherto undescribed.

In conclusion the authors state that a diagnosis of lethargic encephalitis must be reached by exclusion, but that a combination of any two or more of the following symptoms should suggest encephalitis lethargica: (1) fever at the onset of the illness; (2) an acute mental disturbance; (3) a gradually increasing lethargy; (4) a clinical picture of paralysis agitans; (5) myoclonic movements; (6) neuritic pains, which may alternate from side to side; (7) ocular palsies; (8) pupils which do not react to light.

POTTER, Mercer, Pa.

ENCEPHALITIS LETHARGICA. A. J. HALL, *Lancet* **202**:526 (March 18) 1922.

Hall reports thirty cases of lethargic encephalitis, and divides them into three groups: (1) Five mild or abortive cases of which only one, with myoclonic contractions of the abdominal muscles, is of interest. (2) Fourteen cases of medium severity. One was a boy of 12 years, who, on the nineteenth day of his illness, when convalescence had apparently been established, developed suddenly a series of left sided seizures, with loss of consciousness and a temperature of 105 F., after which he improved rapidly. In ten of these cases recovery was retarded or incomplete, and there developed mental deterioration, varying grades of Parkinsonian complex, or paraplegia. In one, of neuritic type, there was a complete flaccid paralysis of both legs, which began five weeks after the initial lethargy and cranial nerve palsies, and disappeared some eight months later. (3) Eleven fatal cases. In commenting on the after history of sixteen previously reported cases, the author calls attention to two patients who had suffered with a polyneuritic syndrome. One made a complete recovery and the other was still unable to walk after three years. In conclusion Hall remarks that it is often impossible during the progress of a case to form an opinion as to the prospect for recovery.

POTTER, Mercer, Pa.

PATHOLOGICAL FINDINGS IN A CASE OF CHRONIC PROGRESSIVE ENCEPHALITIS. M. G. MARINESCO, *Rev. Neurol.* **29**:1012-1019 (July) 1922.

Marinesco reports in detail the necropsy findings in a case of progressive Parkinson syndrome, in which death occurred two years after the onset of epidemic encephalitis. In this case there had been no interval, after the acute stage, in which there was freedom from symptoms.

The pituitary body was found to be normal. In the suprarenals there was infiltration with plasma cells and lymphocytes around the blood vessels. In the suprarenal medulla, which was involved more than the cortex, the chromatin cells showed alterations. In the parotid gland there were marked inflamma-

tory changes which indicated that infection was still active. Marinesco refers to Netter who has frequently found a similar condition in the parotid, and is inclined to believe that it may serve as a focus for the continued production of toxins.

In the central nervous system, the cortex, corpus Luys, the red nucleus and the nucleus of the hypoglossal nerve showed only slight alterations. In the putamen there were vascular lesions and disintegration of the myelin. In the globus pallidus the cells showed two types of change: (1) a lesion which included both degeneration and atrophy; and (2) one in which the volume of the cells was normal but there was dissolution of the chromatin substance, with pale staining of the nucleus and vacuolization. In the substantia nigra there were marked alterations. The pigmentary cells especially had undergone cytolysis, the nerve fibers were much decreased, and there was plasma cell infiltration and neuroglial proliferation about the blood vessels and throughout the white and gray matter. The floor of the fourth ventricle showed an infiltration as marked as that in the substantia nigra. In the muscles, the nerve fibers showed degeneration and the connective tissue enveloping them had increased.

Marinesco suggests that the muscular changes are secondary to mesencephalic lesions which, by producing functional disturbance in the cerebral ends of the autonomic system, cause faulty metabolism in muscular tissue. This is evidenced not only by the anatomic changes but also by electrical variations in muscle response and in reduced temperature in muscle tissue. Marinesco accounts for the marked degeneration and atrophy of cells in the substantia nigra by an especially poor resistance to cytolytic toxins. He disagrees with Hunt as to the part played by the globus pallidus in paralysis agitans, but does agree that both the globus pallidus and the substantia nigra play an essential rôle in the symptomatology of paralysis agitans and post-encephalitic Parkinson syndromes.

HYSLOP, New York.

ATROPHY OF THE CEREBELLUM IN ADULTS WITH THE REPORT
OF A CLINICAL CASE. ARRIGO FRIGERIO, Riv. di Patol. Nerv. e Ment.
27:457 (Oct. 15) 1922.

The author's patient, a woman of unknown age, was admitted to the hospital on Feb. 26, 1920. Her age appeared to be about 65 or 70. About seventeen years before, she began to have difficulty in walking, which she attributed to the fact that she had to carry heavy weights on her head. Gradually the gait became rolling and unsteady, and occasionally she fell backward. In the next few years the disturbance of gait became so severe that she spent much time in various hospitals. Finally, she was committed by the police, because she went about ringing bells, refused food, and had other mental symptoms.

When examined her speech was slow, scanning and explosive. She omitted the final vowels in words and gave to the dentals the sound of palatals. There was a lateral tremor of the head when it was held upright, which disappeared when she rested in bed. The left pupil was larger than the right, but the reactions were normal. There was horizontal nystagmus in the extreme lateral positions, especially on fixation to the right; on looking upward and downward the nystagmus was rotary. The fundi were normal. When the arms were extended, there were massive oscillations which were not seen when the patient was at rest. The finger to nose test showed dysmetria with oscillations at the end of the movement. All movements were awkward. When the

patient grasped a glass, there was exaggerated opening of the hand. There was adiadokokinesia, especially in the left. There was hypotonia in all joints of the left upper extremity, but the Stewart-Holmes phenomenon was inconclusive. The heel to knee, and toe to elevated object, tests showed marked oscillations but the movements ceased when completed. The patient remained supine with legs and thighs flexed until the observer was fatigued; the author calls this cerebellar catalepsy. The patient often assumed a catatonic attitude, with the head flexed and the chin on the chest, for long periods without fatigue or effort. Extension of the limbs tended to be performed in two motions. Closing the eyes had no effect on the dysmetria. There were no disturbances of sensibility. The deep reflexes were all increased and there was a tendency to patellar clonus. The corneal reflexes were unequally active and the abdominal reflexes were absent. When the patient sat, there was lateral swaying of the trunk and a tendency to slump backward. When standing, the feet were unduly spread, the arms akimbo and the head bent forward. This attitude was accentuated by the kyphosis which was present. If she was not held on both sides, she would immediately fall backwards. However, she could walk forward with assistance on each side or with crutches. The crutches were held unduly separated and it might almost be said that she walked on four extremities. When she walked, the limbs were hyperflexed and the feet were slapped violently to the ground, or the legs were thrust out violently or crossed. The steps were of unequal, irregular length and progression, with lateral titubation. The Bárány and cochlear investigations were negative. There was no vertigo, headache, nor vomiting during the month in the hospital. Mentally, the patient showed mild intellectual dullness with a tendency toward irritability, but she was fairly docile.

The diagnosis made by the author was cerebellar atrophy. He discusses the various types of this condition and uses the classification of Dejerine: (1) Partial or asymmetrical: (a) secondary to foci of softening, and possibly of hemorrhage, but usually follows sclerosis of the cerebellar hemispheres; (b) congenital types. (2) Generalized symmetrical atrophies of: (a) the sclerotic type, secondary to inflammatory vascular reactions; (b) agenetic type; (c) the parenchymatous, degenerative type involving the cells of Purkinje and their fibers. (3) Another type is described by Dejerine and Thomas wherein the cerebellar cortical lesions are associated with degenerations in the pons and the olivary bodies. In some of these cases described by other authors, there were diffuse degenerative changes in the striatum or the frontal lobes, or in the bulb and rubrocerebellar or olivocerebellar connections.

Reference is also made to the *dyssynergia progressiva cerebellaris* of Ramsey Hunt. The author believes that his case belongs to the third sub-group of Dejerine. On the basis of his clinical findings, he speculates on the possible pathology. He eliminates the various pathologic types, and compares his case with that described by Ramsey Hunt.

OSNATO, New York.

THE IMPORTANCE OF A SPECIAL EDUCATIONAL TRAINING FOR
MENTAL DEFECT DEPENDENT UPON ORGANIC LESIONS.
L. PIERCE CLARK, *Ment. Hyg.* 6:708 (Oct.) 1922.

This article urges the importance of differentiating in the matter of treatment between cases of mental defect due to organic lesion and the so-called genetic or hereditary types, since in the former the undamaged portions of the brain are capable of benefit by the same kind of training as the normal brain.

To illustrate, Clark cites three cases in which the mere designation of mental defect was not specific enough to serve as a basis for training and would have resulted in lasting detriment to the child if followed to a final disposal of the case. The first is that of a 6-year old girl suffering from cerebrocerebellar diplegia. She was speechless and had the repellent leer of a low-grade mental defective. Tests showed that the greater part of her apparent mental deficiency was overlaid by the motor defects. Instead of being sent to a school for defectives, she was placed with private tutors, under a system of training especially adapted to overcome her handicaps; with the result that at the age of 12 she has begun to attend common school, is nearly up to grade in classwork, and shows an almost uncanny genius for mechanical work.

In the second case, also that of a girl suffering from cerebrocerebellar diplegia, mentality was retarded four or five years, apparently largely as a result of definite nerve lesion. The child was placed in a school for defectives, but did poorly under the routine training given there, and finally was removed and placed with special tutors and with normal associates. Under this treatment her mental progress has doubled.

The third case is that of a boy who, when first seen at the age of 3, was a low-grade idiot. His mental state was apparently secondary to a meningitis, which in turn was secondary to a double pneumonia from a severe chronic colitis at the age of 18 months. He was placed in one of the best schools for defective children for two years; he made some improvement in conduct, but little mental advance. He could not express a want, knew nothing at all about dressing himself, and spent his time roaming about aimlessly. For the last two years he has been under private tutors and has made remarkable strides in mental development. His physical condition was first attended to, as he was suffering from a severe colitis. Mental and speech training were then begun and patiently continued, in spite of the difficulty of holding his attention for more than a few seconds at a time. At present he can make up short sentences, can understand any request, and displays a rather remarkable memory as to where various articles are kept. He knows all the articles of his clothing, and but for a slight deformity in the index finger of each hand which prevents his using them in buttoning, is able to dress and undress himself. He is healthy and happy, and shows more ability to create his own amusements.

In cases of mental defect accompanied by organic lesion, care must be taken to apportion to each factor its proper rôle in the hindrance of development. For children whose inheritance is much tainted, the possibilities of development are poor even when the brain is only slightly damaged; on the other hand, surprising improvements may be expected in children of untainted parentage suffering from brain injury incurred at birth or early in life, with the exception of hemiplegic patients with or without epilepsy; in this respect a double palsy is much more hopeful than a strictly unilateral palsy.

The training treatment for children with acquired mental defect should be directed partly toward overcoming the structural defects in the brain, but should consist principally in a modification of the usual common school methods. Clark emphasizes the importance of association with normal children on terms as nearly normal as possible. For the child suffering from hereditary mental defect, on the contrary, segregation should be sought for the best interests both of the child and of society.

WILLIAMS, New York.

THE PSYCHIATRIC INSTITUTE-HOSPITAL. LEWIS F. PILCHER, State Hosp. Quart. 8:3 (Nov.) 1922.

The author reviews the reasons for the need of a psychiatric hospital and institute that should have facilities for the treatment of early mental disorders and for medical education and research. He reports the recommendation of the New York State Hospital Development Commission, which resulted in definite legislation and the appropriation of money for this purpose. This psychiatric institute-hospital requires six major departments: (1) administration, (2) outpatient clinic, (3) diagnostic clinic, (4) research and teaching, (5) hospital providing 180 beds for adults and 20 beds for children, and (6) residence for personnel.

The building plan, in order to house these departments, should follow the "H" shape plan, including: (a) the clinic institute wing, (b) the hospital wing, and (c) the laboratory wing connecting (a) and (b).

The administration department, including all offices, should be centrally located on the first floor in the institute wing.

Several entrances to the outpatient clinic are advisable so that quiet patients, disturbed patients and children may be admitted separately. The need for a large and adequately lighted waiting room and several examining rooms is discussed, as well as the value of the appointment system in facilitating prompt examinations and avoiding overcrowding. - Separate rooms for roentgen ray, gastro-intestinal, eye, ear, nose and throat, endocrinology, spinal fluid, Wassermann, dental and psychologic examinations should be in close proximity to the clinic.

The diagnostic clinic should be arranged so that the laboratory, examination and treatment rooms are available for both the hospital and outpatient clinics. The necessities for hydrotherapeutic, electrotherapeutic and mechanotherapeutic work are also located on the first floor.

On the second floor, the laboratory rooms necessary for the research work are placed in the arm connecting the institute and hospital portions. These laboratories include the psychologic, clinical, bacteriologic, chemical and neuropathologic group. The author includes in his plan a large lecture room seating 150 people. This room and rooms for a library and museum are placed on the second floor in the institute wing.

On the second, third and fourth floors of the hospital wing, arrangements are made for six classification dormitories. Day rooms opening on porches are also planned. Adequate provision is made on the entire fourth floor for visiting psychiatrists from the various state hospitals and for the hospital personnel.

The fifth floor of the hospital wing contains the major and minor surgical units and space for work in photography and for a gymnasium with its accessories.

EBAUGH, Philadelphia.

A STUDY IN CONSTITUTIONAL PSYCHOPATHIC INFERIORITY. JOHN W. VISHER, Ment. Hyg. 6:729 (Oct.) 1922.

Visher points out the inadequacy of the older definitions and proposes the following description: "From the psychological standpoint, the constitutional psychopathic inferior presents marked inherent defects in volition and inhibition, together with lowered threshold for and a disproportionate response to implicit and explicit stimuli. There is also a lack of balance in the various hereditary and acquired reaction patterns and habit systems. The individual is unable

to adjust to his inadequacies, either by means of experience or by the formation of compensatory modes of reaction. From the behavioristic standpoint, the condition is characterized by marked egotism, impulsiveness, poor judgment, non-conformity to ethical and social standards, and inability to adjust or to profit by discipline."

A statistical study of fifty psychopathic soldiers before, during and after hospitalization, produced some interesting information. Twenty had a neurotic family history, twenty-eight reached the eighth grade or higher, twenty-five had poor industrial records, twenty-six admitted having been arrested in civil life, six were morphin addicts, two were alcoholic, and five used other drugs to excess. Of seventeen who were married, all but five had had serious marital difficulties. Judged by ordinary standards only three of the entire fifty had somatic conditions of sufficient importance to warrant hospital care. The after work record shows that thirty-two of the patients were employed less than 10 per cent. of the total time since discharge from the hospital.

The author concludes that "military service greatly decreased industrial efficiency and led to prolonged hospitalization" and that "treatment of the adult psychopath by psychotherapy, re-education and hospitalization is of but little avail. Vocational guidance is theoretically indicated, but practically is usually unsuccessful.

STRECKER, Philadelphia.

PSYCHOLOGY IN MEDICINE. F. L. WELLS, *Ment. Hyg.* 6:700 (Oct.) 1922.

Wells attributes the present ambiguous relations between psychology and medicine to medieval dualism, which resulted in their development as two unrelated fields. It is only recently that psychology has become one of the biologic sciences, as the science that has to do with the processes by which the organism reacts as a whole to the environment and to its own conditions, and hence, in one of its aspects, the science of behavior. And it is only recently that medicine has turned from its exclusive preoccupation with bodily processes and begun to assume responsibility for the behavior of the organism as a whole. Meeting thus in the field of human conduct, medicine and psychology have not as yet joined forces, with the unfortunate result that psychologists inadequately equipped with medical knowledge are called on to deal with medical problems, while medical men with little grounding in psychology are confronted with psychologic problems. Two solutions are possible: medicine and psychology can enter into the same cooperative relationship as that now existing between medicine and chemistry, or psychology can develop a new discipline that will to a certain extent compensate for lack of medical affiliations. Wells feels that the former course will better serve the interests of the two sciences as well as of society.

With regard to psychoanalysis, while granting that creditable work has been done by nonmedical psychologists, he inclines to the opinion that psychoanalytic material belongs to the field of medicine rather than that of psychology, and that the practice of psychoanalysis involves risks to both operator and subject that should be reluctantly undertaken outside the setting of responsibility that medicine traditionally provides.

Again, in the matter of the exercise by psychologists of public functions, especially the commitment of feeble-minded persons, he feels that the responsibility should not be assumed by a nonmedical person, since every case of mental defect is potentially a medical problem. On the other hand, the type of experience embodied in the American Psychological Association's Section of

Clinical Psychology should be represented in such cases, and the committing physician has not always had the opportunity to acquire such experience. For states such as New York, where this type of expert experience is readily available, Wells suggests a law to the effect that one of the committing officers shall have had training of this type.

The protection of the public from inadequately trained psychologists must, he feels, ultimately rest with the public itself; but scientific psychology may properly be asked to formulate adequate standards and to make service governed by such standards reasonably available. The regulation of consulting psychologists by the general association, and the Psychological Corporation, are moves in this direction.

WILLIAMS, New York.

SOME OBSERVATIONS ON QUALITATIVE CHEMICAL AND PHYSICAL STIMULATIONS IN NUDIBRANCHIATE MOLLUSKS WITH SPECIAL REFERENCE TO THE ROLE OF THE "RHINOPHORES."

H. P. KJERSCHOW AGERSBORG, *Jour. Exper. Zool.* **36**:423 (Nov.) 1922.

An investigation was made to determine the exact function of the dorsal tentacles of three species of mollusks. These tentacles have come to be considered as organs of smell, and are generally called "rhinophoria." *Hermisenda opalescens* Cooper responds to tactile stimuli applied to any part of the body: the heads, the oral and dorsal tentacles, the body, the various parts of the foot, and the papillae. The dorsal tentacles give the most effective response to a tactile stimulus, such as the end of a glass rod. The head and the dorsal tentacles are most sensitive to acids and salts in solution. The tentacles are more sensitive, the tips giving the most effective response. The oral tentacles are almost as sensitive to stimuli as are the dorsal tentacles, but in addition to a general response of this nature, the oral tentacles also have a selective function in that when they are stimulated by some palatable food, the animal may be made to move in the direction of the stimulus; if the animal responds to a stimulus negatively, the appliance of it in front of a progressively moving animal may bring it to a halt; the directions of its movements may also be changed. The oral tentacles have the power of discrimination between certain substances, such as food and odorous oils, while the dorsal tentacles lack this power for the same substances. There is no evidence that the "rhinophores" are olfactory in function.

The dorsal tentacles of *Dendronotus giganteus*, like those of *Hermisenda*, are the most sensitive parts of the body to tactile stimuli. In *Melibe leonina* Gould the cirrhi are more sensitive to tactile stimuli than are the dorsal tentacles.

WYMAN, Boston.

ON THE OCCURRENCE OF REFLEX ARCS IN THE MYENTERIC AND SUBMUCOUS PLEXUSES. ALBERT KUNTZ, *Anat. Rec.* **24**:193, 1922.

Synaptic relationships are described which involve an axon in a commissure which connects the submucous with the myenteric plexus and a neuron located in a ganglion in the latter plexus. The synapses involve two neurons and constitute a nervous mechanism within the walls of the stomach and intestine through which local reflexes may be carried out. Fibers which arise primarily in the submucous plexus terminate in the muscularis mucosae among the glands in the mucosa, and also in the gastric and intestinal epithelium. The fibers which terminate in the epithelium are probably the dendrons of

neurons in the submucous plexus. The findings afford a structural basis for the rational interpretation of all the usual activities of the stomach and intestine except the rhythmic contractions which, according to the work of Magnus, Gunn and Underhill, and Alvarez and Mahoney, are of a myogenic nature.

NIXON, San Francisco.

THE NEED OF PSYCHOPATHIC DEPARTMENTS IN STATE HOSPITALS. JOHN R. ROSS, *State Hosp. Quart.* 8:34 (Nov.) 1922.

Psychopathic hospitals have an important place in the prevention and cure of mental diseases. Ross states that the establishment of psychopathic departments in state hospitals will play an important part in the care of early mental disorders, when the patient is most susceptible to remedial treatment. These psychopathic units should be known throughout the district as places where advice and treatment can be obtained without judicial commitment to a hospital for the insane. The author rightly feels that with such a unit properly manned, treatment of patients would improve and real curative measures be introduced. The article is concluded with a discussion concerning building plans, arrangements for classification of patients, personnel, nursing care, laboratory facilities and occupational therapy. The need of the outpatient clinic and the part such a unit should play in public education regarding mental diseases, is emphasized.

EBAUGH, Philadelphia.

ALCOHOLIC PSYCHOSES BEFORE AND AFTER PROHIBITION. HORATIO M. POLLOCK, *Ment. Hyg.* 6:815 (Oct.) 1922.

Pollock uses the term alcoholic psychosis as defined in the federal census report on the insane in hospitals in 1910: An alcoholic psychosis is one of the mental diseases that are known by their characteristic symptoms to be the direct result of alcoholic intemperance; cases of mental disease in which alcoholic intemperance is only one of the etiologic factors and cases merely associated with alcoholic intemperance should not be classed as alcoholic psychoses.

Using the 1910 census as a basis for comparison, the present study gives the results of various compilations of data on alcoholic psychoses since that census was taken. These data show that there has been a marked reduction in the prevalence of alcoholic psychoses throughout the United States since 1910—a reduction due partly, Pollock believes, to restrictions on the liquor traffic and partly to changes in the habits of the people. Of 60,769 patients with mental disease admitted to institutions in 1910, 10.1 per cent. were reported as cases of alcoholic psychosis, while the annual rate of admission of alcoholic cases was 6.7 per 100,000 of general population. The latest available data concerning the prevalence of alcoholic psychoses throughout the country were compiled by the statistical department of the National Committee for Mental Hygiene from original standardized reports of state hospitals. Based on the statistics from the hospitals of fourteen states, the percentage of alcoholic admissions to all admissions in the year 1921 was 3.1 and the rate of incidence in the general population 1.9, a noticeable decline from the census rates.

A corresponding decline appears in the figures for the New York state hospitals in the years 1909-1921, as compiled by the statistical bureau of the New York State Hospital Commission. In 1909, the percentage of alcoholic first admissions to all first admissions was 10.8, and the rate of incidence in the

general population was 6.3; in 1921, these rates were 2.8 and 1.8, respectively. At the same time, there has been a decline in the number of cases among all first admissions in which there is a record of intemperate use of alcohol; in 1909, such cases constituted 28.7 of all first admissions, while in 1921, this rate had declined to 11.8. Data from the official records of Massachusetts tell the same story: in 1912, the percentage of alcoholic first admissions to the state hospitals for mental disease and McLean Hospital was 11.3; in 1921, it was 4.9.

In all these compilations, the lowest rates appear in the year 1920. The slight increase in 1921, Pollock attributes partly to lax administration of the liquor laws, partly to economic depression.

That the rate of first admissions with alcoholic psychoses is closely correlated with the per capita consumption of liquors is evident from a comparison of the index numbers of rates of alcoholic first admissions to New York state hospitals with the index numbers of per capita consumption of liquors in the United States for the twelve years from 1909 to 1920. The coefficient of correlation between the two series is 0.875 ± 0.045 . Pollock is of the opinion that an even closer correlation would have been found had the data relative to the per capita consumption of liquors in New York state been available.

Comparative statistics from twenty states with regard to the sex distribution of alcoholic first admissions for the years from 1919 to 1921 show that the rate of decline under prohibition has been relatively greater among women than among men. The suggested explanation is that women are less likely than men to come in contact with the bootlegger.

During the whole period for which adequate data are available, alcoholic psychoses have been much more prevalent in urban than in rural districts. In 1910, the rate of alcoholic admissions in the United States was 10.7 per 100,000 in the urban population and 2.6 in the rural, and the admissions from urban districts constituted 77.8 per cent. of all the alcoholic admissions. This percentage has increased since. A study of first admissions to the New York civil state hospitals from July 1, 1915, to June 30, 1920, showed that 90.9 per cent. of the alcoholic first admissions were from urban districts. These figures are borne out by representative data compiled by the statistical department of the National Committee for Mental Hygiene from the state hospital reports of several states in 1919, 1920 and 1921. In these years, the percentages of alcoholic first admissions from urban districts were 85.6, 83.9 and 84.4, respectively, while the percentages from rural districts were 14.4, 16.1 and 15.6.

WILLIAMS, New York.

A QUANTITATIVE STUDY OF TARSAL SENSITIVITY TO SOLUTIONS OF SACCHAROSE, IN THE RED ADMIRAL BUTTERFLY, *PYRAMEIS ATALANTA* LINN. DWIGHT E. MINNICH, Jour. Exper. Zool. **36**: 445 (Nov.) 1922.

Many insects are extremely sensitive to distant chemical stimulation. The nymphalid butterflies, *Vanessa antiopa* and *Pyrameis atalanta*, possess taste organs or contact chemoreceptors on their ambulatory tarsi. Distilled water and solutions of several substances, including molar saccharose, can be differentiated through the tarsal organs. Contact of the four ambulatory tarsi with molar saccharose solution will always effect an extension of the proboscis, and so will contact with distilled water after a prolonged period of inanition with respect to water. The latter response ceases if the animal is allowed to drink water. By administration of water, *Pyrameis* can be maintained in a state of 0 per cent. responsiveness to water, but of 100 per cent. responsiveness to molar

saccharose. The minimal concentration of saccharose necessary to effect a response can be determined. In *Pyrameis* the threshold of response to saccharose solutions varies directly with the nutritional condition of the individual. During periods of total inanition followed by periods of water diet, that is, during saccharose inanition, it gradually falls. But with the initiation of a period of saccharose diet, it rises abruptly to a level which remains constant throughout the remainder of the period. After prolonged inanition with respect to saccharose the threshold concentration may fall as low as M/3,200, M/6,400, or even M/12,800 in some individuals. The tarsal sensitivity of *Pyrameis* to saccharose may thus be as much as 256 times that of the human tongue. The highly developed sensitivity to saccharose is doubtless correlated with the fact that sugars form the chief food of this insect.

WYMAN, Boston.

THE GENETIC ORIGIN OF DEMENTIA PRAECOX. FREDERICK W. MOTT.
J. Ment. Sc. **68**:333 (Oct.) 1922.

Mott's conception of dementia praecox is rigidly organic, and he has ingeniously linked his original studies of testicular degeneration in this disease with a fairly well supported premise of the inadequacy of more recently acquired brain structure. The neocortex consists of six cellular layers: one molecular, three pyramidal, one granular and one polymorphous. The psychomotor cells of earlier ontogenesis may be regarded as belonging to a lower evolutionary level, and their axonic myelization is older than that of other neurons. The supragranular layer is a later acquisition than the infragranular, both phylogenetically and ontogenetically. The former is associated with educability as contrasted with the reproductive and vegetative functions of the latter. The neocortex as a whole represents the final, highest and most complexly differentiated evolutionary structure, and as such is peculiar to homo sapiens. Following the biologic dictum of Hughlings Jackson "The last cerebral structures to come evolutionally and developmentally are the first to go." Mott suggests that dementia praecox, or primary dementia as he prefers to call it, may be referred to a genetic inadequacy which is partially manifest in the reproductive organs at adolescence, and which reveals itself clinically through an arrest of neocortical growth. Physical, physiologic or psychologic stresses or strains may act as precipitating agents.

"I am of the opinion that all the psychoses belong to one group and are genetic in origin. In those forms of psychoses in which recovery takes place—for example, confusional insanity or exhaustion psychosis, benign stupor, periodic insanity, or manic-depressive insanity—we may assume there is a suspension of neuronic function in the highest psychic level; but all these conditions I have found may end in terminal dementia, in which the changes in the reproductive organs and in the brain do not differ from those met with in the primary dementia of adolescence, the dementia indicative of a suppression of function. In the primary dementias, naturally, the symptoms may be partially due to a suspension, and partially to a suppression of function, and I would explain partial remission of symptoms by a partial restoration of function in neurones in which the nuclear change was either not present or not advanced."

The foregoing statements are interesting, but somewhat startling. For the present, they must be dismissed with the old Scotch verdict "not proven," with the sincere hope that Mott and his co-workers will continue their investigations.

STRECKER, Philadelphia.

BODY AND MIND: THE ORIGIN OF DUALISM. FREDERICK MOTT, *Ment. Hyg.* 6:673 (Oct.) 1922.

Mott reviews briefly the various stages in the history of philosophical and scientific thought that led from the primitive conception of the body and the soul as separate entities—a conception centering largely around the idea of death—to the modern view of the inseparability of mental and physiologic processes.

From the standpoint of biologic psychology, the sources of mental activity in man and animal are the three primal instincts: self-preservation, propagation and the herd. The physiologic bases of these instincts are specific biorhythms that in the course of ages have become fixed and organized in the bodily structures, especially the nervous system, in response to particular stimuli that subserve these instincts. Associated with the primal instincts are certain states of feeling and bodily reactions that constitute the primitive emotions. These instinctive reactions can take place independently of consciousness; Mott cites cases of decerebrate dogs that manifested anger and disgust. The intimate relationship between these instincts and emotions and the structures of the body is evidenced by the profound influence of the glands of internal secretion, especially the sex glands, on mental life.

Any experience attended by deep feeling and emotional discharge sets up in the unconscious sensorimotor and vegetative systems the physicochemical changes that constitute the biorhythm attuned to the particular emotion involved. Long after an experience with an intense emotional tone has left the field of consciousness, it may still be operating subconsciously, the involuntary and unconscious bodily expression of the emotion exciting the associated subconscious memory of the experience and this in turn reacting on the body.

The vicious circle thus established explains (1) the perseveration of hysterical derangements of mind and body resulting from emotional shock, and (2) anxiety neuroses involving a conflict between the instinct of self-preservation and the herd instinct. In the late war, cases of the former were cured by breaking the vicious circle through cure of the bodily symptoms by various methods of contrasuggestion or persuasion; a complete change of mental attitude followed bodily recovery.

In the anxiety neuroses, the form of nervous disability most common among officers, the instinct of self-preservation played a less dominant rôle than among the rank and file, the anxiety neurosis having its origin in fear of losing the esteem of the herd. The fact that the mental and physical effects were frequently similar to those produced by fear for self-preservation may be explained on the ground that the two instincts have been attuned by evolution to a corresponding biorhythm in respect to fear.

The article closes with a short description of Hughlings Jackson's three evolutionary levels.

WILLIAMS, New York.

MIXED CELL SARCOMA OF THE BRAIN. LEO M. CRAFTS, *J. A. M. A.* 79:1910 (Dec. 2) 1922.

At the age of 22, the patient was struck on the forehead by a plank. Following this he suffered from headache, and later mental changes were noted. A diagnosis of brain tumor in the right frontal region was made, and three operations were performed at intervals of two months. At the second and third operation a tumor mass or masses were removed from the right frontal lobe. By reference to the roentgenograms and skull measurements, it appears that

one tumor passed through 1 inch (2.54 cm.) and the other through 2 inches (5.08 cm.) of brain substance spontaneously to reach the surface. After two years, there was no indication of further recurrence. Another interesting feature of this case was the presence of well marked speech disturbances in a right-sided lesion; the patient began to write with the left hand, but was trained to use the right.

NIXON, San Francisco.

SOME VARIETIES OF TRAUMATIC AND TOXIC ULNAR NEURITIS.

E. F. BUZZARD, *Lancet* **202**:317 (Feb. 18) 1922.

In this paper the author invites attention to neuritis with no obvious anatomic features, cases with additional toxic factors and those with some anatomic peculiarities. Numerous cases illustrating each group are cited briefly. In the first group, excessive or prolonged use of the arm or hand in a fixed position is considered as the chief exciting cause. In the second group some infective or septic illness is held to be an added factor. In the third group Buzzard places those cases with a history of a severe injury to the elbow at some time, as well as cases without the history of such trauma but with some deviation from the normal anatomic relations. In this last group he describes several cases in which the ulnar groove was shallow and permitted the nerve to slip over the inner condyle, and thus to be exposed to more or less continuous slight injuries, which resulted in the formation of a fusiform neuroma with constricting fibrous bands. In such cases he advocates the transposition of the nerve to the front of the elbow and the removal of the constricting bands. This author believes that a careful examination should suffice to distinguish ulnar palsy from spinal cord disease or from cervical ribs resulting in plexus lesions.

POTTER, Mercer, Pa.

ON THE AFFERENT PATHS OF THE SYMPATHETIC NERVOUS SYSTEM, WITH SPECIAL REFERENCE TO NERVE CELLS OF SPINAL GANGLIA SENDING THEIR PERIPHERAL PROCESSES INTO THE RAMI COMMUNICANTES. OTTORINO ROSSI, *Jour. Comp. Neurol.* **34**:493, 1922.

Since Kölliker's anatomic demonstration in 1894 of fibers passing from spinal ganglions into the sympathetic nervous system through rami communicantes, there has been accumulating a large amount of evidence that these fibers form the chief (if not the only) pathway for sensory nervous impulses from the sympathetic to the cerebrospinal nervous system. But no one has hitherto pictured the actual connection of these fibers with cells of the spinal ganglions. This Rossi has accomplished in Golgi preparations of the bird (sparrow) and mammal (pig). These neurons are regarded as afferent visceral sensory elements.

C. J. HERRICK, Chicago.

MUTUAL RELATIONSHIP IN THE DEVELOPMENT OF THE BRAIN AND EYES OF LEPIDOPTERA. STEFEN KOPÉČ, *Jour. Exper. Zool.* **36**:459 (Nov.) 1922.

The whole brain (ganglion supra-oesophageale) was removed from caterpillars of *Lymantria dispar* L after their last moult, and the caterpillars were allowed to develop into adults. The larval optic ganglions were also removed in some. The eyes of the moths developed in complete independence of the brain.

and the subesophageal ganglion. The brain exerts only a regulating influence on the direction of the nerve fibers going from the retina of the eye to the optic ganglion. On the other hand, if the imaginal eye, the germ of which had been removed in the caterpillar, is absent, the external layers of the optic ganglion do not develop, and certain internal layers show changes in their structure. The germs of mature eyes grafted from the head of the caterpillar on its abdomen develop normally, notwithstanding the absence of any junction with the nervous chain. In specimens deprived of brain the subesophageal ganglion develops to a markedly less degree than in normal specimens. The removal of the larval subesophageal ganglion has no visible effect on the formation of the imaginal brain.

WYMAN, Boston.

NEUROPATHIC ARTHRITIS. JOHN H. DUNCAN, J. A. M. A. **79**:1987 (Dec. 9) 1922.

The case reported represents what happens to a joint when afferent nerves are destroyed. The patient received an injury to the right shoulder, in which the posterior roots of the lower cervical and upper thoracic roots were divided or torn out. There was complete disorganization of the carpus, and it was impossible to recognize all the carpal bones. The distal ends of the radius and ulna were hypertrophied, and the periosteum of each showed bone formation. From the clinical and roentgenographic appearance of the joint, the increased density with the absence of pain, the associated area of anesthesia and the history of injury, the author concluded that the condition was a neuropathic arthritis.

NIXON, San Francisco.

HYSTERIA. A. BALDIE, Dublin J. M. Sc. **4**:529 (Dec.) 1921.

Baldie defines hysteria as "state of mind depending upon a restriction of sentiments and emotions, resulting in a moral perversion of the individual, due to a failure of adaptation to environment, characterized objectively by the occurrence, as rationalizations, of phenomena which are defensive or protective in origin." This author believes that a diagnosis of hysteria depends primarily on the presence of the philomorbic syndrome: a defect in character evidenced by the restriction of the sentiments, an antipathy to activity, and a disordered suggestibility based on this. An organic basis, for the protean objective phenomena encountered, must be excluded. In considering the differential diagnosis, Baldie calls attention to the importance of distinguishing between neurasthenia and hysteria. He states that neurasthenia is a condition which is due to an intense or prolonged psychologic conflict, while the hysterical person represents the type of psychoneurotic who has solved his conflict in the wrong way. Another source of error, in making a diagnosis of hysteria, is the presence of an incipient organic nervous disease. Given the necessary environment for a cure, the prognosis in hysteria is good. The stimulation of inhibited or absent sentiments or desires and their conversion into habits of mental activity is essential. Voluntary congenial and profitable work is also necessary.

POTTER, Mercer, Pa.

BRACHIAL MONOPLÉGIA DUE TO THROMBOSIS OF THE SUBCLAVIAN VEIN. GEORGE WILSON, Am. J. M. Sc. **163**:899 (June) 1922.

A survey of the literature shows the relative rarity of subclavian thrombosis. Wilson points out that in none of the cases cited was a brachial

monoplegia secondary to the phlebitis. The two cases presented by the author occurred in young negroes. Both gave a history of sudden onset. They awoke to find the hand and arm tense and swollen, and the edema persisted for some time. A complete monoplegia was at once apparent, but gradually disappeared as the swelling of the arm subsided. In one case, however, return of function occurred only above the elbow, the muscles of the forearm showing atrophy and degeneration. The other monoplegia improved steadily, due perhaps to the multiple incisions made in the skin to reduce the edema at the time of the thrombosis.

Syphilis is generally considered to be the etiological factor, though one case has been reported during typhoid fever. One of the author's cases was definitely syphilitic, the other presented a clinical picture of tuberculosis, which was later verified at necropsy. As far as can be determined thrombosis of the subclavian vein occurs very rarely in tuberculosis. The monoplegia which results is not permanent, but must be differentiated from a cerebral monoplegia with an associated vasomotor paralysis.

TEMPLE FAY, Philadelphia.

GLIOMA OF THE CEREBRAL HEMISPHERES, A COMPARATIVE STUDY OF TWO CASES. TOM BENTLEY THROCKMORTON, J. A. M. A. 79:1913 (Dec. 2) 1922.

In both cases reported, the cardinal symptoms of increased intracranial pressure—headache, vomiting and choked disk—were latent features; and it was not until the ventricular current was interfered with through pressure that the general symptoms became evident. In one case, the sudden onset and prodromes of an infectious process simulated epidemic encephalitis; in this case, there was a slowly developing motor paralysis resulting in total hemiplegia and seemingly due to a vascular lesion until pressure symptoms developed. In the second case, the early clinical picture was not unlike that of beginning paresis.

NIXON, San Francisco.

SPINAL FLUID CHANGES FOLLOWING AIR INJECTION. G. HERMANN, Med. Klin. 18:1146 (Sept. 3) 1922.

Hermann reports several cases in which a spinal fluid examination was made before and after injection of air. In a case of general paralysis the first examination showed 16 cells; fourteen hours after the injection of air the cell count was 2,231. This rapidly declined and by the fourteenth day the count was 19. A few weeks later following a second air injection, the number of cells mounted to 754. In a case of dementia praecox with a cell count of 2, there was, following air injection, a count of 11,400, chiefly polymorphonuclear leukocytes. Clinically, this patient showed a meningitis as indicated by the temperature, headache, vomiting and a positive Kernig sign. Five days later there was marked improvement with a cell count of 100.

The author feels that air injection is not without danger. The reaction is probably due to stimulation of the meninges. It is interesting that the reaction is less marked when there is disease than in cases with supposedly normal meninges. Hermann does not wish to be dogmatic as more work of this kind must be done. He thinks it is barely possible that the injection of air may serve as a therapeutic measure, especially in cases of general paralysis and tuberculosis. It, in some measure, resembles the production of pneumoperitoneum in tuberculous peritonitis. The use of gases and narcotic substances

might be suggested, but this must be carried out with extreme care in view of some of the disastrous results which have followed lumbar anesthesia.

MOERSCH, Rochester, Minn.

OCCUPATIONAL THERAPY AS A MEANS OF CONTROLLING DISTURBED PATIENTS. WALTER G. RYON, *State Hosp. Quart.* **7**: No. 3 (May) 1922.

Ryon states that the object of occupational therapy is for the sublimation of the activities of the disturbed patient from a pathologic channel to a normal one which gives satisfaction in place of the usual destructive tendencies. This direction of the energy of the patient should not, in the author's opinion, be limited to occupational classes alone, but should include the work of the hospital in any department. Recreation of all types is recommended. Occupational therapy should be used not only in the treatment of the acutely disturbed patients, but also in caring for the chronically disturbed patients who show marked habit deterioration. This work is accomplished by classes of habit training using simple games, marching to music, gymnasium and kindergarten work, wood-sawing, sand-papering, etc. The success of habit training in other state hospitals is referred to, especially the work of Dr. Read in Illinois. The author concludes his article with five abstracts of cases showing the value of occupational therapy in bringing the patient into closer contact with reality.

EBAUGH, Philadelphia.

A CASE OF RETROBULBAR NEURITIS ASSOCIATED WITH SYMPTOMS AND SIGNS OF DISSEMINATED SPINAL SCLEROSIS. H. ARMSTRONG, M. J. *Australia* **2**:22 (Nov. 26) 1921.

The author presents a meager case history and evidently reached his diagnosis by exclusion. The patient when 22 years of age, suffered a loss of power in both legs, and some anesthesia in the same region. Recovery occurred in six months. Several years later he temporarily lost power in the left shoulder. At 35 he complained of heaviness of both lower limbs and a numbness in the arms and legs. On examination both patella jerks were exaggerated and there were numerous scattered patches of analgesia over the inner aspects of both arms and legs; speech was slow and slurred but not scanning in type. There were characteristic signs and symptoms of retrobulbar neuritis. The administration of potassium iodid in doses of 0.6 gm. resulted in recovery. In discussing the case, Armstrong reviews its various possibilities and concludes that there must have been an association of retrobulbar neuritis with inflammatory patches in the spinal cord.

POTTER, Mercer, Pa.

NEUROPATHIC ARTHROPATHIES—CHARCOT'S SPINE. J. RIDLON and E. J. BERKHEISER, *J. A. M. A.* **97**:1467 (Oct. 28) 1922.

The authors report ten cases of tabetic Charcot's spine. This condition may occur in any part of the spine, but in their experience it was confined to the lumbar spine. Of their patients, only one was a woman. The ages at which the condition was noted varied between 32 and 68 years. The average number of years between the initial infection and the occurrence of the arthropathy was eighteen years. The bony changes show bone destruction, bone proliferation and displacement, usually laterally. Traumatism may accenuate the condition. From the examination of the roentgenograms, it would appear

that there are two types of pathologic changes in these joints: the osteo-sclerotic or the consolidation type, and the osteoporotic or the rarefaction type. For spinal support, they advise a plaster, leather or celluloid jacket made over a corrected plaster torso.

NIXON, San Francisco.

PACHYMEMINGITIS HEMORRHAGICA INTERNA: A STUDY OF FIVE CASES OF NON-TRAUMATIC HEMORRHAGIC SPINAL FLUID. ARTHUR D. DUNN, *Am. J. M. Sc.* **163**:819 (June) 1922.

The author presents five cases that show a striking similarity of symptoms and from which he draws a diagnostic triad. The condition, which is usually associated with syphilis, was found mostly in persons past middle life. One case, however, was in a girl aged 18. Four of his cases gave negative Wassermann reactions.

The combination of a bright red, uniformly bloody cerebrospinal fluid in cases of sudden intense headache with signs of meningeal and root irritation, establishes the diagnosis. The symptoms common to all cases were: Headache, intense, sudden in onset, usually occipital or at the base of the neck; rigidity of the neck; deep hyperesthesia; Kernig's sign; paravertebral tenderness; muscle stiffness and soreness; increased reflexes and Babinski signs. Lumbar puncture relieved the intensity of the headache and this procedure was repeated well into convalescence. The fluid obtained became brown and finally yellowish as the patient improved. Of interest was the fact that, with recurrence of the attacks of severe headache, fresh blood reappeared in the spinal fluid. The persistence of muscle cramps, fatigability, instability, restlessness and insomnia into the late period of convalescence was characteristic of all cases.

TEMPLE FAY, Philadelphia.

EXCESSIVE GOLF FOLLOWED BY MUSCULOSPIRAL PALSY. C. C. WHOLEY, *J. A. M. A.* **79**:2,000 (Dec. 9) 1922.

The patient complained of severe pain to the left of the spine over the lower part of the neck, which would come on as soon as he lay down. There was weakness of all the extensor muscles of the left upper extremity. The man gave a history of an active life, and of being an enthusiastic golf player.

NIXON, San Francisco.

IS THERE A BABINSKI SIGN OF THE HAND ? A. RADOVICI, *Presse méd.* **30**:767 (Sept. 6) 1922.

The case described and illustrated here was that of a quadriplegic with marked automatic movements in all four extremities. Pinching the skin, from above the umbilicus to the upper edge of the clavicle, induced triple flexion of the corresponding arm. The palm-chin reflex (of Marinesco and Radovici) was absent. Stimulating the palm, particularly the thenar eminence, with a needle, caused extension of the thumb and fanlike spreading of the fingers. This movement was slow and quite similar to the great toe sign of Babinski.

In this case there was fracture of the atlas and axis with excessive callus, so that the lesion was just high enough to avoid causing a flaccid paralysis of the arm. In ordinary hemiplegia, automatic movements do not occur in the upper extremities, and the "Babinski sign of the hand" is not found.

HUDDLESON, New York.

THE NORMAL SELLA. C. D. ENFIELD, J. A. M. A. **79**:934 (Sept. 16) 1922.

From a study of the roentgenograms of the sella turcica in 100 consecutive cases, excluding cases of definite endocrine disturbance, the author concludes that the sella turcica in the normal person may vary both in size and in contour within very wide limits; that the average sella, which has usually been interpreted from the roentgen-ray findings as being the normal type, constitutes in the neighborhood of 50 per cent. of the general run of cases, and that wide variations from the average are not necessarily accompanied by any indications of pituitary malfunction or disease. He believes that the only definitely and positively abnormal roentgenographic finding is clear evidence of erosion of the bony structures.

NIXON, San Francisco.

MENTAL FACTOR IN VISCEROPTOSIS. W. H. B. STODDART, *Lancet* **1**:69 (Jan. 14) 1922.

The author calls attention to the common association of anxiety states, visceroptosis and mobile right kidney. In explanation of the mechanism, Stoddart says that any anxiety or fear state produces an excess of epinephrin, which stimulates the gastric sphincter and results in food retention and gastric fermentation, which finally causes gastropptosis with secondary visceroptosis. He believes that all patients with anxiety neurosis and hysteria should be examined for evidence of visceroptosis.

POTTER, Mercer, Pa.

THE TREATMENT OF RESIDUAL EPIDEMIC ENCEPHALITIS.

GEORGE H. HYSLOP, *J. Neurol. & Psychopath.* **3**:250, 1922.

Fifteen cases of the parkinsonian type of encephalitic residua were studied in relation to their reaction to drugs. With the exception of opium, the only results of value were secured from the administration of scopolamin or gelsemium, the former yielding improvement in 75 per cent. of the cases and the latter in 66 per cent. Two patients seemed to recover under the influence of gelsemium.

SINGER, Chicago.

PSYCHOSES ASSOCIATED WITH SYDENHAM'S CHOREA. E. M.

HAMMES, *J. A. M. A.* **79**:804 (Sept. 2) 1922.

In a series of eighty-eight cases of Sydenham's chorea, the author noted definite symptoms of a psychosis in eighteen of the patients. The mental picture showed considerable variation, except that frequently distressing hallucinatory ideas were present. The mental picture conforms to that observed in toxic psychosis.

NIXON, San Francisco.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Stated Meeting, Dec. 5, 1922

FOSTER KENNEDY, M.D., *President, in the Chair*

CASES ILLUSTRATING SOME ASPECTS OF BRAIN AND SPINAL CORD TUMORS. CHARLES A. ELSBERG, M.D.

Case 1. The first case was a patient on whom laminectomy had been performed, with removal of an extramedullary leiomyoma at the level of the second and third cervical segments, secondary to a tumor of the neck which had been removed eighteen months previously. The man, a civil engineer, aged 53, was admitted to the Neurological Institute Jan. 2, 1922. For a number of years he had suffered with neuralgia of the left side of the face, which was relieved by the extraction of some of his teeth. In 1915, he had a small growth on the right side of the neck, which slowly increased in size and was accompanied by pain in the back of the neck. In 1920 he was operated on by Dr. John Worcester of New York. At this time, the tumor was as large as a hen's egg, and was situated in the anterior triangle of the neck on the right side. At the operation, the tumor was found to be underneath the large vessels; it was adherent to the deep muscles of the neck and lay against the transverse processes of some of the cervical vertebrae. The tumor was well encapsulated and in one spot seemed to extend into an intervertebral foramen. The pedicle extending into the foramen was ligated and cut, and the entire growth was removed. The pathologic diagnosis was a leiomyoma of branchiogenetic origin.

A few months before this operation the patient began to suffer from occasional severe pain in the back of the neck on the right side, which radiated up into the head. Six months after the neck operation, he noticed that the right index finger was becoming weak, and the weakness gradually involved the entire hand and arm. One week later, there was beginning loss of power in the right lower extremity. About this time the left lower extremity, and later the left upper extremity, became weak. In August, 1921, laminectomy was performed in the lower cervical and upper thoracic region but no tumor was found. The patient did not improve after the operation, and his symptoms steadily progressed. The pain in the neck which was continuous, became very severe, so that he could not move his head in any direction. At times he had an exquisitely cold sensation in the hands and feet, especially on the right side. The weakness in the limbs gradually became worse and he was bedridden for two months, with almost complete loss of power in both upper and both lower extremities. For a month before admission to the hospital he had incontinence of urine and feces.

When admitted to the hospital, physical examination revealed a tall, strongly built man who was considerably emaciated; he was delirious at times and complained of great pain in his neck. The right pupil was slightly smaller than the left, but both pupils reacted promptly to light and accommodation:

the fundi were normal, although the retinal veins were somewhat overfilled. In the right upper extremity power was absent at the shoulder, elbow and wrist. Slight flexion and extension of the fingers were possible, but the movements of the index finger were slight. In the left upper extremity, slight abduction and adduction at the shoulder, and slight flexion and extension at the elbow were possible; good power was present at the wrist; flexion of the fingers was fair, but extension of the fingers was weak; pronation of the forearm was weak, and supination of the forearm was absent. The pectoral reflexes were weak on the right, normal on the left. The biceps and triceps reflexes were markedly exaggerated on both sides. The radial and ulnar reflexes could not be elicited on the right, and were weak on the left. There was complete loss of power in the abdominal muscles and the cremasteric reflexes could not be elicited. The right lower extremity was decidedly weaker than the left, and the power in both limbs was greatly diminished. The suprapatellar, patellar and Achilles reflexes were weak on both sides, and a Babinski reflex was present on both sides.

The disturbance in tactile, pain and temperature sensibility involved the entire body, from the second cervical segment downward. Disturbance in joint and vibratory sensibility involved the greater part of the body. There was marked tenderness on pressure over the upper cervical spinous processes.

Roentgen-ray examination was negative. Lumbar puncture produced a clear fluid containing six cells to the cubic millimeter. The globulin reaction was +++; the Wassermann test was negative.

Jan. 7, 1922, a laminectomy was performed, with removal of the arches of the second, third, fourth, and part of the arch of the fifth cervical vertebrae. Incision of the dura exposed a well encapsulated tumor, which was firmly adherent to the dura, and about 3 by 2 cm. in size. It was situated at the level of the second cervical segment, ventrolateral in position, and on the right side. Into it ran the third cervical root. The tumor had a projection which extended through the dura at the level of the intervertebral foramen between the second and third cervical vertebrae, but there was no evidence of tumor on the outside of the dura. The third cervical root on the right side was divided inside the dura and the tumor was removed entire with its capsule. The cord had been pushed markedly to the left and was much flattened; the arachnoid was distended above and below the tumor.

Improvement in the power of the upper limbs was noted by the patient as soon as he awoke from the anesthesia, and the return of power and sensation was so rapid that in three weeks he was able to move the right upper extremity with almost normal force at the shoulder, elbow, wrist, and all finger joints. The power in the right lower extremity was also greatly improved. The left upper and lower limbs could be moved with normal strength in all directions. There was a corresponding improvement in the sensibility of the body. The joint sense disturbances had not improved, but vibratory sensibility had returned in both lower limbs. The patient left the hospital less than seven weeks after the operation. At that time he walked freely without any support, and could use both upper extremities freely and well. The cutaneous sensation over the body and extremities was only slightly disturbed. He has since steadily continued to improve.

The examination of the tumor removed from the patient's neck in 1920, was reported as follows: "The tumor measured 4 by 2 by 2 cm. and was well encapsulated; it was moderately firm in consistency, and on gross section showed mottling of light yellow with grayish-white translucent areas. Micro-

scopically, the principal elements were smooth muscle cells, closely packed in bundles, and fat cells of the embryonal type; there was a moderate amount of fibrous stroma; the cells were uniform in size, did not exhibit anaplasia and no mitotic figures were seen. Diagnosis: leiomyoma."

The intradural growth was also well encapsulated; it showed at one end a knob-like projection where it had extended through the opening in the dura for the third cervical root. Microscopically, the growth was the same as, but more vascular than, that of the tumor removed from the neck.

This was a very unusual case of a growth which began in an intervertebral foramen and grew outward between the deep structures of the neck. When the tumor of the neck was removed, a ligature was placed around its pedicle, which extended into an intervertebral foramen. The scar tissue which resulted, prevented a recurrence into the neck, and the growth then extended inward, through the opening in the dura for the third cervical root, and formed an intradural tumor in that location.

Case 2. Dr. Elsberg presented a patient on whom he had operated for extradural fibrosarcoma, and who had completely recovered. The patient, a girl, aged 16, was admitted to Mt. Sinai Hospital on April 19, 1921, with a history of pain for two years on the left side of the chest, behind and beneath the angle of the scapula. The pain became so severe that for a time she required large doses of anodynes. Two months before admission, her left leg became weak and stiff, and a few weeks later the right lower extremity became similarly affected; she also noticed that the feeling in the right lower extremity was not as good as formerly. The weakness and stiffness of the lower limbs, and the diminution in sensibility of the right leg, grew rapidly worse, so that the patient could walk only with great difficulty and could not stand without support. For one month she was unable to empty her bladder, and a catheter had to be used.

Physical examination showed that the abdominal reflexes were very weak on both sides. The left lower extremity was much weaker than the right. The knee jerks were much exaggerated; there were: double patellar clonus; double, inexhaustible ankle clonus, and a Babinski sign on the left side. The fifth thoracic spine was very tender, especially when pressure was made on it from the left. There was a marked diminution of tactile, pain and temperature sensation on the right side below the fourth thoracic segment, and a sensory disturbance on the left.

The roentgen-ray examination was negative. Lumbar puncture produced a clear fluid with 5 cells to the cubic millimeter; the globulin was not increased and the Wassermann reaction was negative.

On April 11, 1921, a laminectomy was performed, with removal of the arches of the fourth, fifth and sixth thoracic vertebrae. On the posterior and left side of the dural sac, and adherent to it, was a well encapsulated tumor measuring about 4 by 1 cm. The tumor was of firm consistency, not very vascular, and did not involve any of the nerve roots. It was removed in several pieces together with part of the transverse process of the fifth thoracic vertebra and a piece of the dura. Pathologically, the tumor was a fibrosarcoma which contained a large number of foreign body giant cells.

After the operation there was rapid improvement. Complete control of the bladder was regained within one week. All sensory and motor symptoms disappeared within six weeks. For several months the patient complained of slight pain in the back, but this gradually disappeared. She received roentgen-ray treatment at regular intervals. Eight months after the operation she had

gained 20 pounds in weight and felt perfectly well. Eleven months after the operation roentgen-ray examination revealed a small shadow to the left of the area of the former operation. This shadow had increased in size. There were no subjective or objective neurologic disturbances. An incision was made through the old wound which exposed and permitted the removal of a bony tumor, which was evidently callus, from the region of the former operation. There was no evidence of recurrence of the growth. Pathologic examination of the tumor showed newly formed bone with no evidence of fibrosarcoma. At the present time the patient is free from all symptoms.

Case 3. Dr. Elsberg presented a boy who had been admitted to the service of Dr. F. Tilney at the Neurological Institute in January, 1922. He had been perfectly well until May, 1921, when he had an attack of pain in the right side of the chest which extended upward into the head. In June, he had a second attack and with this a sensation as if his right arm was contracting. Between these attacks he had minor seizures of the same nature. During the following month he had four severe seizures, all beginning with a pain in the lower part of the thorax. The case has been fully reported by Dr. H. A. Riley in the *Neurological Bulletin*. The signs pointed to a tumor in the left superior parietal lobule; at the operation a large angiosarcoma, 5 by 6 cm. in size, was removed from the superior parietal lobule, behind the Rolandic fissure. The tumor was well encapsulated, but was not clearly differentiated from the cortex in the deeper areas. The patient is now almost free from symptoms. Except for one convulsive attack he has been well since the operation.

Case 4. Dr. Elsberg presented a patient from whom he had removed a large endothelioma of the anterior part of the left temporal lobe in August, 1922. The tumor had given rise mainly to mental disturbances and subjective sensations of numbness in the left hand. The patient recovered completely and has remained well.

Case 5. Dr. Elsberg also presented a left-handed man, from whom he had removed a large tumor of the right frontal lobe eighteen months before, with very satisfactory recovery.

In connection with these cases Dr. Elsberg presented a number of very large brain tumors, both cortical and subcortical, which had been removed during the past year from patients on the service of Dr. Tilney at the New York Neurological Institute. All of these patients recovered from the operation.

DISCUSSION

DR. F. TILNEY: One point to be noted is the large number of endotheliomas in this group of cases. This is surprising if we remember that they represent a collection of cases which have occurred within the last twelve months. It is rather unusual to see such a number of surface tumors which are accessible. The general tendency of this group is to affect certain parts of the brain, namely, the frontal and prefrontal areas. In the case with acrognosis and astereognosis there was a typical cortical anesthesia. The growth itself extended back into the parietal lobe, but the involvement was mainly of the frontal and prefrontal areas. I would like to speak of the group method employed in the diagnosis which has proved to be of very distinct value. Each case was thoroughly presented and discussed by the five men who had studied its various phases. We did not use ventriculography, for the results without that procedure were satisfactory in regard to localization.

DR. FOSTER KENNEDY: In the frontal tumor case, was the blindness in the left eye due to pressure on the optic nerve?

DR. F. TILNEY: Some years before symptoms began this man was examined for fitness to do motorman's work. There was found, at that time, a defect in the left eye, so that this symptom should not be attributed to the neoplasm. He was rejected for motorman's work after that examination. He is left-handed, which, I think, explains the fact that there was no aphasia.

DR. M. A. STARR: In the neurologic examination of the elderly woman, was there any appreciable mental change, such as loss of memory, or slowness of thought, in connection with the development of pressure on the right frontal lobe?

DR. F. TILNEY: There was considerable mental disturbance prior to the operation. That was the most outstanding feature of the case. The patient had loss of memory for recent events and some obscuring of remote memory. After the operation there was marked euphoria. This gradually subsided. Her husband says that since the operation she has undergone a complete change in disposition. Her memory has returned and she has been able to carry on efficiently the management of her household.

STUDIES IN FOCAL INFECTION: ITS PRESENCE AND ELIMINATION IN THE FUNCTIONAL PSYCHOSES. NICHOLAS KOPELOFF, PH.D., by invitation, and C. O. CHENEY, M.D.

The conclusions from this study of the relation of focal infections to functional psychoses may be summarized as follows: (1) The removal of infected teeth and tonsils from twenty-seven cases showing manic-depressive, dementia praecox, and psychoneurotic reactions, has been followed by no more mental benefit than was observed in a comparable group of thirty-three patients from whom such hypothetical foci of infection were not removed. There were no recoveries or distinct improvements other than those to be expected, irrespective of focal infection. (2) The Rehfuess method of fractional gastric analysis is not to be relied on as a means for determining gastric infection. The bacteria found in the stomach contents by this method may be derived largely or entirely from the swallowed saliva. (3) Chronic constipation may be relieved by means of milk fermented by bacillus acidophilus, reinforced with lactose. (4) These studies are being continued, not only for the purpose of obtaining further facts about focal infection in the psychoses, but as part of a general plan to afford psychotic patients every available opportunity for benefit.

DISCUSSION

DR. HENRY A. COTTON: The work of Drs. Kopeloff and Cheney bears a striking resemblance to ours at Trenton in 1916, and the results are what might be expected. In the first fifty patients whose teeth were extracted and tonsils removed we obtained no results. There are two reasons for this: first, the work was limited to the teeth and tonsils; second, we selected our cases and were disappointed in the results. It was not until 1918 that we began to examine every case admitted in a systematic, routine manner. At the end of 1918, we found we had produced results. The authors have had our experience to guide them, but apparently have not benefited by that experience. I have talked this matter over with them, but they seem to ignore the facts and insist on considering that their work in a way offsets ours of the last four years. The error in their work is evident to anyone who gives

the matter consideration. In the first place, in very few patients, especially in women, is the infection limited to the teeth and tonsils. Eighty per cent. of the women have gynecological infection, mostly in the cervix, and at least 50 per cent. of both sexes have secondary foci in the stomach which must be treated by vaccine or specific serum.

Dr. Kopeloff claims to have made a very exhaustive investigation of gastric infection and is inclined to dispute the results obtained by Rehfuß and by us. The principal point in dispute is the origin of the bacteria found in the stomach. He makes the claim, from what I think is insufficient evidence, that the bacteria found in the stomach by the Rehfuß method come entirely from the saliva which is swallowed when the test is made. In order to decide this point we made cultures from the saliva in the mouths of 106 patients at the same time that the stomach test was made. In only fourteen cases did we find any relation between the bacteria in the saliva and the bacteria in the stomach. That is, in these fourteen cases, the bacteria in the two localities were identical and were limited to one type of streptococcus. In the other ninety-two cases, entirely different strains of streptococci were found in the mouth as compared with the stomach. In some cases the same strain was found in both, but there were other strains as well. This tends to disprove Kopeloff's contention without doubt, and also to substantiate the work of Rehfuß.

There seems to be little reason for this report. Twenty-five cases are entirely too few on which to base an opinion; and the work which has been done on them has been entirely inadequate. Such work does not explain the success of the methods at the State hospital at Trenton. In the last four years 1,400 cases have been treated successfully, and have been considered as recovered when they were discharged. Of this number only sixty-two have been returned and are now in the hospital. The question might be asked: How do we know that these cases, some of them after four years, are still well? The answer is simple for the patients are visited regularly by field workers and reports are obtained at least twice a year. These patients have been thoroughly detoxicated and all infection has been eliminated by surgery, where possible, and, in addition, by autogenous vaccines, and antistreptococcic and anti-colon serums.

It is easily demonstrated from our work that the mere extraction of a few teeth and the enucleation of a few tonsils will not produce results. In this so-called functional group, our records show that, for a period of ten years, 37 per cent. recovered spontaneously. In the last four years the recovery rate in the same group, where treatment has been energetically applied, averages 85 per cent. These are facts which cannot be controverted.

We do not include in these 1,400 patients, 250 who have left the hospital unimproved. The majority of these were received from other institutions and the psychoses had been of such long duration that nothing could be done for them. These patients frequently return to the institutions from which they came and, of course, serve as an object lesson for the poor work done at Trenton. At least six State hospitals are following our methods and are obtaining similar results. While it is disappointing to have this report from Ward's Island, we nevertheless, feel that it in no wise decides the question.

DR. G. H. KIRBY: The revival of interest in the study of the physical condition of patients suffering from mental and nervous disorders is an important forward step in psychiatric work. It cannot be denied that in some quarters at least there has been a tendency to neglect the somatic aspect of

mental disorders while the psychogenic viewpoint has been pushed to the front. The idea that nervous and mental affections of the functional type are due primarily to some bodily disorder or toxemia is, of course, a very old one in psychiatry. But up to the present there have been few, if any, well controlled scientific observations which tended to confirm this hypothesis. Aside from a very small group of psychoses, practically nothing has been established to show a definite etiologic relationship between toxemia and mental disturbance. In much larger groups of mental and nervous affections the same cause has been rather widely assumed to exist. Such assumption, however, was based almost solely on empirical observations and the apparent effect on patients of certain methods of treatment. The nature of the problem has made the proving or disproving of etiologic relationships a very difficult matter. Naturally, a wide interest was at once aroused in recent claims that the problem had been solved; that there was no essential difference between the clinical groups of dementia praecox, manic-depressive and the psychoneuroses; that they all have a common and a relatively simple cause—namely an infection in some part of the body—which leads to a slow poisoning of the central nervous system, with the result that neurotic and psychotic symptoms appear. It has been claimed that a mild toxemia produces a mild or benign psychosis (manic-depressive or psychoneurosis), whereas a severe or prolonged toxemia leads to dementia praecox.

On grounds of clinical experience it is difficult to accept such a generalization, which sweeps away the whole structure of clinical distinction based on symptomatology, course and outcome of these mental affections, and ignores all that has been established regarding the rôle of personality and psychogenesis in functional nervous and mental disorders. One must, therefore, insist on a careful scientific working out of the questions at issue under conditions in which chances for error are reduced, the permanency of results is established, the personal equation is eliminated, and a comparable control material is carefully studied. Dr. Kopeloff's communication is a preliminary report on the way in which the work is being conducted at the Psychiatric Institute. Our purpose has been merely to state our findings after a careful study of the problem up to the present time. A larger clinical material is being investigated and a report will be given later.

DR. M. OSNATO: What is our attitude to be when these patients have foci of infection?

DR. F. H. DIETERICH (by invitation): What is the proportion of infected teeth in the general admissions to psychiatric institutions? And what is the cause of the insanity when there is no focal infection?

DR. C. E. GIBBS (by invitation): I have been closely associated with Dr. Kopeloff and am familiar with his work. It is necessary to establish definitely what constitutes a focus of infection outside of the teeth and the tonsils. It remains to be demonstrated that bacteria free in the stomach are a source of infection or intoxication. Are they any more a focus of infection than are those free in the mouth, in the saliva? Dr. Cotton stated that 80 per cent. of his female patients have infection of the cervix. One of Dr. Cotton's associates recently stated at a meeting that any cervical discharge meant a focus of infection. I do not think that belief will be generally accepted.

DR. G. H. HYSLOP, by invitation: Dr. Cotton stated that infection of the cervix is very common in the psychoses of women. I heard Dr. Maxwell say that the bacteria found in the cervix originated in the tonsils, and that they reached the cervix through the blood stream and lymph channels. They

might go by the blood but I do not see how they could reach the cervix by the lymph paths. The most direct way would be along the intestinal canal. It is really remarkable how very few clear-cut functional psychoses result from infection. This does cause delirium or mental confusion, but the number of manic-depressive or dementia praecox cases is negligible. It may be that infection precipitates an attack. We have seen a number of Dr. Cotton's patients, many of whom have returned with the handicap of lack of teeth. In regard to erosions of the cervix, it is remarkable how many women have erosions and discharges; yet relatively few of them develop psychoses. I believe that the value of Dr. Cotton's work is that he has called attention to the mouth, tonsils, and colon; surely stomach infection is rare. Prior to his work the teeth and mouth were utterly neglected, but I do not think that all his claims are justified.

DR. L. PIERCE CLARK: The issue at stake in Dr. Kopeloff's presentation is much broader than the question whether a focal infection was a causative factor in the type of cases presented here, or even in the more extensive work of Dr. Cotton, previously reported. The question arises whether the psychoneuroses and psychoses are caused by focal infections or by any other type of infection. Does their etiology rest on such a cause as is popularly stated in textbooks and taught in the medical school, or have they a causation on more fundamental biologic or psychobiologic principles, such as makeup, and difficulties in life adaptations? Dr. Cotton, in particular, refuses the latter any place in etiology, and places emphasis on purely somatic and local infective agents. I believe such a view is wrong; it runs counter to all conceptions of psychogenesis and mental pathology. These rest on too firm a scientific foundation to be thus uprooted. The whole movement of focal infection in psychoneuroses, while in the immediate interest of good internal medicine in mental hospitals, is a regressive tendency as it gives mental pathology a secondary place in the domain of psychiatry.

DR. KOPELOFF, closing: It is hardly fair for Dr. Cotton to say that our results are identical with those he obtained in 1916, if for no other reason than that we have no detailed report of those results. It is true that in some of our patients examined by Dr. Cotton there were tonsillar remnants. This is not unusual, even in Trenton. The tonsillar remnants were removed after Dr. Cotton saw these patients and yet their condition has remained the same. Dr. Cotton claims that bacteria in the stomach cause a low gastric acidity, but a very careful examination of his tables in an article recently published in the *American Journal of Psychiatry* (October, 1922), reveals the fact that streptococci were found irrespective of high or low degrees of acidity. With regard to his attempts to disprove the claim that bacteria in the stomach come to a large extent from the saliva, his technic is open to severe criticism. We plated out the bacteria quantitatively at once. Dr. Cotton puts a sample in broth and then incubates. This permits one kind of organism to predominate, and one cannot tell on the following day what types of organisms had been present on the day before. Our criticism of Rehfuess rests on two grounds: first, that the gastric contents are not homogeneous, and second, that a single fractional gastric analysis is insufficient ground on which to base a conclusion as to the gastric acidity. Gorham, Wheelon, and White have furnished additional evidence along these lines. With regard to operative procedure, we did not neglect operations when necessary, but we did not operate without adequate indication. Dr. Cotton remarked that we cannot cure colon lesions with acidophilus milk. We did not claim this, but stated that we could relieve

chronic constipation by this means, and this removes Dr. Cotton's cardinal symptom for abdominal surgery. Dr. Hyslop and Dr. Gibbs have already discussed infection of the cervix as it was presented last month by Dr. Langstroth. It is sufficient to state that he claims to cure, not *only* functional psychoses by enucleation of the cervix, but also epilepsy and even feeble-mindedness. In answer to the question whether or not one should eliminate focal infections in psychotic patients, I believe we agree that all infections should be removed. However, what influence this will have on the psychosis is another matter. That, indeed, is the reason for our work.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Dec. 21, 1922

E. W. TAYLOR, M.D., *in the Chair*

CASE OF CEREBELLAR TUMOR: OPERATION: RELIEF OF SYMPTOMS. DR. E. W. TAYLOR.

This case is presented because of the purity of the cerebellar symptomatology. Apart from certain signs of general increased intracranial pressure, there are absolutely no indications of cranial nerve paralysis or other neighborhood symptoms. The patient was well so far as he knows until three months ago. He then had a certain difficulty in the management of the left leg with a slight tendency to limp and to turn toward the left in walking. A week after the onset he noticed clumsiness of the left hand. These signs increased in severity. He was admitted to the Massachusetts General Hospital about two months after the onset. For several days previously he had vomited excessively, for the most part irrespective of food, and often in a projectile manner. He had also had considerable vertigo, which he at times somewhat vaguely described as headache. He was mentally clear during this period. The physical examination at the hospital revealed normal reflexes and sensation. The optic disks were slightly clouded. He was much disturbed by a sense of vertigo and occasional vomiting. There was horizontal nystagmus in both eyes on lateral fixation. His strength was unimpaired, but there was very marked disturbance in the left side, characteristic of cerebellar asynergy. Dysmetria, adiodokinesia, Holmes' rebound phenomenon in less degree, and past pointing toward the left, were all markedly present. He became unable to walk without assistance, and when helped had a strong tendency to go toward the left. He stood as well in the Romberg position with open as with closed eyes. The sphincters were at no time involved and he remained mentally clear.

The diagnosis of left cerebellar hemisphere lesion was made, presumably a tumor, possibly a cyst. Operation by Dr. W. J. Mixter, November 29, after exposure of both cerebellar hemispheres revealed very marked pressure with particular bulging on the left side. Incision of the hemisphere led to the evacuation of a considerable amount of yellowish fluid. Palpation revealed a tumor which proved to be gliomatous in character. After the operation the patient improved considerably in his general symptoms from the relief of pressure. The cerebellar signs, however, persist, but in somewhat less degree.

Treatment by roentgen-rays may relieve the situation, but the prognosis is obviously grave.

An unusual feature of the case is the absence of choked disk in spite of extreme intracranial pressure. The rapidity of development of the tumor is also noteworthy, and is probably to be explained by a secondary cyst formation.

DISCUSSION

DR. W. J. MIXTER: The tumor in this case was seated deeply beneath the cortex of the cerebellar hemisphere, and was associated with a cystic cavity. The tumor was soft, friable, and impossible to remove. Under microscopic examination it proved to be a glioma.

DR. H. C. SOLOMON: I would like to ask what is the value of roentgen rays in the treatment of a case of this type.

DR. W. J. MIXTER: I am not enthusiastic about the effect of roentgen rays in this case. In general, however, I am convinced that it helps in many instances, particularly in the soft, embryonic type of tumor.

CHRONIC LEAD POISONING VERSUS TABES DORSALIS. DR. GEORGE CLYMER.

This case is one of long-standing disability which has given rise to considerable difference of opinion as to its cause. The patient is a man aged 55, who was in the Massachusetts General Hospital from Oct. 22, 1919, to Dec. 8, 1919. His occupation for thirteen years was that of a sign painter, but for ten years he was unable to work, and during that time there was no history of exposure to lead. The past history contains a record of an accident at the age of 20 in which his right knee was crushed; since then it has been weak. There was a venereal discharge for two weeks sixteen years ago but syphilis is denied. His wife has had two miscarriages and there are no living children.

He complained of: (1) Abdominal pain, located in the region of the umbilicus, two to three times a week; it came on ten to fifteen minutes after meals and was gnawing in character. It would disappear for three to four months at a time and then return more severely. (2) Dizzy attacks without loss of consciousness about once a month in which he fell to the floor, but recovered after a minute or two. (3) Pain in the extremities of ten years' duration, with a tingling sensation in one foot and leg, and pain "like being squeezed in a vise" in the other leg. This pain was constant, but there were periods of more severe pain which lasted two and a half hours, and occurred at first every two to three weeks, recently every three to four days. (4) Dyspnea and palpitation of the heart without edema, for ten years; orthopnea at night. Eight years ago he was told that he had heart trouble, and ten years ago that he had lead poisoning. (5) A stabbing precordial pain with a sensation of numbness in the left arm which began four months ago. Belching of gas, constipation and nausea for the past two months.

While in bed in the hospital the dizziness disappeared but the other symptoms, especially the abdominal pain, became worse. He also said that he had had a dry cough for months, and had noticed that his voice was growing hoarse.

Physical examination at the time of admission revealed a larger pupil on the right than on the left; both were regular and reacted to distance but not to light. Many teeth were missing. There was no lead line on the gums.

The spine presented a double right scoliosis. The apex impulse of the heart was seen and felt in the fifth space; the rate was slow and there was an occasional extra systole; the sounds were of poor quality and there was a late systolic murmur; the second aortic sound was not heard; the first sound at the apex was sharp and loud; there was no diastolic murmur. The pulses were equal and of good tension. The artery walls were palpable. In the abdomen there was a suggestion of a mass in the epigastrium; liver dulness extended from the fifth space to 5 cm. below the costal margin; the edge was felt three finger-breadths below the costal margin and was not tender. The right knee presented a very resistant swelling and the patella was dislocated toward the right. The knee jerks were absent; there were neither clonus, nor Babinski or Kernig signs. Through the rectum a large, boggy but not tender prostate was felt.

The laboratory findings were: Blood Wassermann test negative. Lead positive in the urine. Functional kidney tests: October 24, 10 per cent.; October 25, 25 per cent.; October 28, 40 per cent.; October 31, 5 per cent.; November 3, none; November 23, 60 per cent. The blood count revealed: white cells, 4,400; red cells, 3,972,000. In blood smears no stippling was seen in three examinations; the red and white cells appeared normal. The nonprotein nitrogen was 54.32 mg.; creatinin, 1.3 mg. The stools contained lead. The spinal fluid was clear and under normal pressure; it contained no cells and protein and globulin tests were negative. The Wassermann reaction was negative. The roentgen-ray examination was entirely negative. A bismuth enema revealed an extra loop in the descending colon, but no other abnormality.

During his stay in the hospital on that occasion, he was seen by eleven consultants from the surgical and neurologic departments, the department of industrial medicine, and the department of syphilis and gastro-enterology. The opinions recorded varied from a positive diagnosis of lead poisoning to a positive diagnosis of tabes dorsalis, and a diagnosis of combined lead poisoning and tabes.

He has now returned to the hospital with a definite Charcot knee. The other symptoms are much as they were in 1919. He came to the hospital now because while walking, on the day before admission, he felt as though something had slipped in his knee, and he has been unable to bear weight on it since. The laboratory findings now are: The blood Wassermann is negative; lumbar puncture yielded 10 c.c. of clear fluid under normal pressure which contains 6 cells; the alcohol precipitate is positive, the ammonium sulphate negative; total protein, 44 mg.; the goldsol reaction is 0123321000; the Wassermann reaction is negative. The urine is negative. Blood examination reveals: polymorphonuclears 59, lymphocytes 36, large mononuclears 5; the red cells are slightly achromatic; the platelets are normal. Stool examinations are negative.

Roentgenograms bear out the clinical diagnosis of Charcot knee and the lancinating pains which he has now are characteristic of tabes.

It now appears that the laboratory tests for lead which were made at the time of his previous admission would be positive not only with lead but also with manganese, so that the laboratory reports that lead was present cannot be taken as conclusive. In the absence of other evidence of lead poisoning, or of exposure to lead within the past ten years, it is probable that lead can be ruled out as a factor in the clinical picture at that time. The serologic findings then and now are characteristic of a relatively inactive stage of tabes in which Charcot joints frequently develop. These cases unfortu-

nately respond little if at all to antisypilitic treatment. At the suggestion of Dr. J. B. Ayer, the patient has been given sacral injections of 65 c.c. of physiologic saline solution which have relieved the pain.

This case emphasizes the point that it is easy to be led astray by laboratory findings. On his first admission they apparently demonstrated the presence of lead and the absence of syphilis. We now believe that lead was probably not a factor in the case, and that the picture was due entirely to syphilis.

HYOSCIN IN PARKINSONIAN RIGIDITY. DR. GEORGE CLYMER.

This man, aged 22, was suddenly taken sick three years ago with headache, fever, sleepiness, weakness, dizziness, and diplopia. He was then in bed for three days. The symptoms gradually passed off, but in the course of a month or two it gradually became difficult for him to take deep breaths. Eighteen months ago he noticed a slowness of all movements, although there has at no time been any mental retardation.

When first seen here he presented a typical picture of Parkinson's disease, with even a slight tendency to drool. He was admitted to the hospital for serologic and other examinations, which resulted thus: The urine was negative. The blood was normal and the blood Wassermann reaction negative. On lumbar puncture 10 c.c. of clear colorless fluid was withdrawn which revealed cells 2; ammonium sulphate test negative; alcohol test negative; Wassermann test negative; goldsol test, 0000000000; total protein, 45 mg.; pressure normal; sugar 0.0625 mg.

The treatment has been: December 16 and 17, hyoscin hydrobromid, $\frac{1}{150}$ grain (0.0004 gm.) subcutaneously three times a day; December 18 and 19, hyoscin hydrobromid, $\frac{1}{100}$ grain (0.0006 gm.) subcutaneously three times a day; December 20, hyoscin hydrobromid, $\frac{1}{100}$ grain (0.0006 gm.) by mouth three times a day. As a result he now shows no rigidity whatever, and his movements are normal. He also feels well. I do not think that permanent benefit has been derived from the treatment, but offer the case simply to show the palliative results which may be obtained.

BROWN-SEQUARD PARALYSIS FROM DISLOCATION OF CERVICAL VERTEBRA. DR. HENRY R. VIETS.

A boy, aged 16, struck his head on the bottom when he dived into a shallow pool, and was stunned but not unconscious. Immediately and for some weeks afterward he suffered from severe, sharp, and intermittent pains in the neck, upper part of the back, and arms, which caused him many sleepless nights. He could not raise his arms above his head, and the movements of the hands were awkward and clumsy. His fingers, as well as the lateral aspects of his thighs, were numb. Micturition was not affected. He walked with his head forced forward in a semi-flexed position and was unable to turn it to the right or left. Roentgenograms in the anterior-posterior position were taken before he entered the Massachusetts General Hospital, but failed to reveal fracture or dislocation.

When seen at the hospital two months after the injury, he carried his head in the position described. No definite dislocation was noted in the anterior-posterior roentgenograms, though the space between the fourth and fifth cervical spines was increased without lateral displacement. The lateral plates, however, showed a forward displacement of the fourth on the fifth cervical vertebra, without fracture. The pupils were equal. The triceps and radial

reflexes were active and equal, but the biceps jerk could not be obtained; the whole of both arms, except the hands, showed some weakness. Both legs were slightly spastic, with both knee jerks increased, bilateral ankle clonus, and suggestive bilateral Babinski signs. There was also some swaying in the Romberg position. Questionable hypesthesia of patchy distribution was demonstrated over the upper third of the left thigh.

At the operation by Dr. W. J. Mixter, three and one-half months after the injury, bilateral forward dislocation of the fourth on the fifth cervical vertebra was disclosed, with pressure on the spinal cord by the laminae of the fourth. The vertebrae were strongly fused and could not be moved laterally by manipulation, nor did traction change their relation. Laminectomy of the third and fourth cervical vertebrae was done. Now, three months after the operation, the patient's condition is greatly improved, his head is straight and can be flexed and rotated, at least in part, without pain. He has a partial Brown-Séquard paralysis; the right side of the body is partly anesthetic, especially for pain and temperature sensibilities, up to the level of the fourth cervical segment, and the line of demarcation is fairly sharp. On the left side there is spasticity of the leg, with increased reflexes, ankle clonus, and a Babinski sign. The arms have no definite sensory loss, though there is some paresthesia of the fingers. Both deltoids and both biceps muscles are weak, but they respond to faradic stimulation and have slightly improved.

The Brown-Séquard paralysis clearly points to a lesion in the fifth cervical segment of the spinal cord, more definite on the left than on the right side. This localization also corresponds to the paresis of the arm muscles. The diaphragm does not seem to be involved, and this suggests that the fourth segment was not seriously injured. The Brown-Séquard paralysis is of the usual incomplete type, with paresis on the side of the lesion, associated with spasticity and loss of muscle and joint sense, and a contralateral loss of pain and temperature sense. There is diminution of the sense of touch on both sides.

In studying the roentgenograms I lay special emphasis on the value of the lateral exposure as essential to the diagnosis of the lesion. Roentgen-ray examination made before the patient entered the hospital failed to reveal the dislocation because it was limited to the anterior-posterior field.

DISCUSSION

DR. W. J. MIXTER: This case presents one very definite lesson: the danger of spinal cord surgery when moderate pressure is already being exerted on the cord. I used the utmost care in removing the upper laminae which were pressing against the cord, and yet I increased the paralysis very much.

Another point is that after a dislocation of the neck has existed for some months, bony union takes place to such an extent that it is impossible, except by actually cutting the bodies apart, to reduce the dislocation. This is obvious in the roentgenogram in which the bony union is apparent.

VERTIGO AND ITS TREATMENT. DR. C. L. WOOLSEY.

Vertigo is the result of stimulation of the vertiginous centers in the cerebrum through the vestibular end-organ, its tracts, or associated tracts. Vertigo is of two types: subjective, when the individual experiences a sensation of posture change in relation to the objects about him; and objective, when the objects seem to change their position in relation to the individual.

It may be accompanied by a disturbance in the emotional realm or by giddiness, nausea and vomiting. It is essential to ascertain exactly what the patient means by "being dizzy"; frequently, visual blurring is mistaken for vertigo. Vertigo may be caused by injuries to the vestibular end-organ or to its tracts.

CASE 1.—Mr. S. came to the neurologic department of the Massachusetts General Hospital on account of attacks of severe vertigo accompanied by nausea and vomiting. Several weeks previously he had fractured the base of the skull and had profuse bleeding from both ears and from the nose. After several weeks he returned to his former position as locomotive engineer, but experienced severe vertigo and vomiting when taking curves. The neurologic examination was negative with the exception of tinnitus in the left ear and a left horizontal nystagmus. The fundi were negative.

The treatment in this case began by rotating the patient morning and night in the horizontal plane. Three complete rotations of the body were followed by rest in bed; each successive day the number of rotations was slowly increased, so that the patient learned to accommodate himself to the stimulus producing the vertigo. After six weeks treatment he was able to resume his duties as locomotive engineer and since has experienced little or no inconvenience from vertigo. The rotation treatment should not be attempted if the ocular fundi indicate increased cerebral pressure. Only chronic cases should be treated by this method.

CASE 2.—Margaret N., aged 58, came to the clinic complaining of severe objective vertigo. There was no history of head injury. For several weeks the patient had experienced severe attacks of vertigo accompanied by nausea, vomiting and an extreme emotional upheaval. Neurologic examination revealed a rotatory nystagmus with a synchronous head movement identical in direction with the eye nystagmus.

The roentgenogram of the teeth revealed several root abscesses. These teeth were removed and within four weeks the vertigo, nausea and vomiting had ceased.

CASE 3.—G. O., aged 35, was healthy until the age of 27, when she developed pulmonary tuberculosis. She was confined in a state hospital for tuberculosis for one year. During her treatment at this hospital she developed vertigo with nausea and vomiting. Soon thereafter she noted a horizontal movement of the head. The vertigo had persisted intermittently and this brought her to the neurologic department of the Massachusetts General Hospital. Physical examination indicated an active tuberculous process in the apex of the left lung.

The neurologic examination was negative, with the exception of left horizontal nystagmus accompanied by horizontal head movement synchronous with the eye nystagmus. The fundi were negative and lumbar puncture revealed no increase in intracranial pressure; consequently the patient was treated by rotation. After eight weeks she feels that she has been greatly benefited.

CASE 4.—J. J. C., aged 12, came to the clinic on account of severe dizziness, accompanied by nausea and vomiting, which had existed for two weeks. The first symptoms were gastro-intestinal pains and diarrhea. This was soon followed by vertigo, nausea and vomiting. The nausea was increased by a sudden change of position. The patient experienced a sensation of falling forward and to the right. At the time of examination no abdominal symptoms were noted.

The pupils were round, equal, and reacted to light and accommodation. The fundi were negative. There was a slow pull of the eyes upward and to the

right with a resulting quick nystagmus downward and to the left. A movement of the head was noted, identical and synchronous with the eye movement.

This patient was treated with phenobarbital, $\frac{1}{4}$ grain (0.016 gm.), three times a day. Within twelve hours he improved, and within one week he was entirely free from vertigo.

CASE 5.—A. R., a young man aged 33, came to the clinic because of attacks of vertigo. For the past six years he has experienced severe attacks of vertigo, nausea, and vomiting, at first at infrequent intervals. Lately the attacks are so frequent and severe that the patient is unable to earn his livelihood. The patient has a typical Raynaud syndrome, which involves both hands and feet. When this is most pronounced his vertigo is most severe. The pupils are round, equal and react normally. The ocular fundi are negative. At times there is diplopia and a right rotary nystagmus. Tinnitus and some deafness of the right ear are present. Caloric tests indicate interference in the right vertical canals or their tracts in the pons.

This patient has been treated by rotation treatment, phenobarbital and other drugs, with little or no improvement. Since he has been given sodium nitrite, 1 grain (0.06 gm.), three times a day, he has experienced less vertigo.

DISCUSSION

DR. J. B. AYER: Is the rotation treatment original with you?

DR. C. L. WOOLSEY: I know of no one else who has used the rotation treatment. I have communicated with men who have used douching with great relief. If the lesion is on one side only, rotation is contraindicated because it would stimulate both end-organs.

THREE CASES OF FOCAL EPILEPSY. DR. W. J. MIXTER.

The three cases to be described, which have come to operation in the past three months, presented certain strikingly similar features. The condition is not uncommon but is well worth keeping in mind. The histories in brief are:

CASE 1.—Baby S., aged 8 months, was seen with Dr. Bronson Crothers. Delivery was difficult and forceps were used. The infant was apparently normal until 4 months of age. Since then he has not been well; he has seemed dull and has had an increasing number of convulsions which start on the left side of the body. The complete examination, including that of the cerebrospinal fluid, was negative except for slight left facial weakness, and increase in the tendon reflexes on the left side.

CASE 2.—Mrs. L., aged 28, was seen with Dr. F. C. Lord. She gave a long history of attacks of weakness in the right arm, which have not disabled her until two months ago. Since then the attacks have become epileptiform in character and have involved the right side of the body. During the past few days she has been aphasic and has shown definite weakness of the right side of the body. For twenty-four hours attacks have been much more frequent, and now occur at about three-minute intervals. Examination is negative except that: The temperature is 101; the pulse 110; she is unconscious; epileptic convulsions which start in the right side of the face and arm occur every three minutes, last about a minute, and become general after the first few seconds. The left side of the body is flaccid between the attacks. The reflexes vary; they are active at times but are abolished after the convulsions.

CASE 3.—Mrs. D., aged 58, was seen with Dr. H. C. Solomon. For the past four years she has had epileptic convulsions which started in the left arm and

left side of the face and then became generalized. There have been periods of weeks when she was free from convulsions; at other times the seizures have been very frequent. During the past few months there have been observed: definite slowing of her mental processes, forgetfulness, etc. Examination was negative except for: Increased density in the right temporal bone revealed by roentgen ray; slowness and forgetfulness; unsteady gait.

All three cases were operated on, and an osteoplastic craniotomy was performed over the affected area in each case. In all the arachnoid was elevated and the space between the arachnoid and the cortex was filled with clear fluid. The area affected was definitely localized, and in all three cases was anterior to the motor cortex. In Case 2 there was a marked increase in the blood supply to the affected area. Numerous small incisions were made in the arachnoid to permit the fluid to run off, and in Case 2 the enlarged veins were ligated in several places. In all three cases striking improvement followed the operation. Baby S. (Case 1) is a large healthy boy, and is apparently normal except for the scar in his head. Mrs. L. (Case 2) is apparently a normal woman. She has had no attacks since the day after operation and is now, two months after the operation, able to use her typewriter. Physical examination is negative. It is now three weeks since the operation in Case 3. During the first five days this woman had numerous convulsions and it was necessary to turn down the bone flap and remove a thin clot which had formed beneath it. Since that time she has passed through an uninterrupted convalescence and is now far more alert than when she came to the hospital.

The pathologic process in all three cases is difficult to understand. There must be some underlying cause for the collection of fluid beneath the arachnoid, but as yet we have no real information as to the nature of the fluid nor of the underlying cause. It may be that cerebrospinal fluid has collected beneath the arachnoid owing to some fault in absorption of this fluid.

DISCUSSION

DR. E. W. TAYLOR: One of these cases which I had an opportunity to study suggested a purely psychogenic origin for the attacks. What, in general, is the ultimate outcome in cases such as you have described?

DR. S. J. MIXTER: Most of the cases have done well but a good many have recurred. I think that is the usual history. These three are satisfactory thus far but it would be interesting to observe them three years hence; I surmise they would not then be so satisfactory. The second case, alluded to by Dr. Taylor, is of rather peculiar interest on account of the very rapid recovery. I also have a strong suspicion that much of her trouble was functional. In none of these three cases nor in many others has there been a choked disk. One might think of a cortical or dural tumor with pressure on the cortex, but whether choked disk has been present or not, recent cases have led me to hazard a diagnosis of fluid under the arachnoid, whatever its origin may be.

SUBTENTORIAL CYST OF MENINGES WITH NECROPSY. DR. JAMES B. AYER.

Mrs. H. M., Armenian, 23 years of age, was admitted to the Massachusetts General Hospital, Oct. 24, 1922. While in Constantinople, two years previously, she began to be troubled with headache, which increased in severity up to the time of admission. Sixteen months before, she married and came to

the United States, where a healthy full-term baby was born. Since delivery, seven months before admission, bi-temporal and sub-occipital headache have greatly increased; at this time also dizziness set in, and for four months there has been difficulty in walking. For two months she has complained of difficulty in the use of her hands and for two weeks there have been imperative micturition and nasal regurgitation of food. Recently she is said to have been forgetful and to repeat her words.

During the period October 24 to November 21, physical examination revealed: The patient was well developed and well nourished; except for scars of previous variola, pyorrhea alveolaris, and a compensated mitral regurgitation, nothing abnormal was found; the stools showed neither blood nor ova; the urine was negative; the blood Wassermann reaction was negative; there was a slight secondary anemia, but the leukocytes were normal in number and proportions.

Neurologic examination revealed in the cranial nerves: Vision was present in both eyes and there was no marked contraction of the fields as judged by the finger test; perimetry was unsatisfactory from lack of cooperation; the fundi presented bilateral choked disk of 4 to 5 diopters; vertical and horizontal nystagmus were constant; ptosis of the right eye and right sided facial weakness were inconstant and of slight degree; there were no subjective or objective disturbance of sensation of the face nor weakness of the jaws; hearing was present on both sides, but bilateral tinnitus was constant; the ear drums were normal; at times there were difficulty in swallowing and nasal regurgitation of liquids; the gag reflex was absent; the tongue was protruded straight and showed no atrophy nor tremor.

In the cerebral function: there was constant awkwardness in the use of both hands but particularly of the right; hypermetria and asynergy were especially marked in the right hand; no spontaneous past pointing was observed at any time; unfortunately lack of cooperation made Bárány tests unreliable; the gait was unsteady, the right side being especially affected; there was a constant tendency to fall toward the right side.

Sensation was usually normal throughout for cotton-wool, pain, heat, cold, and deep sensibility; on two occasions the reactions to needle pricks suggested hyperalgesia over the whole left side, though tactile sensibility was normal.

The arm reflexes, knee and ankle jerks were equal and lively; at times responses suggestive of Babinski and Oppenheim signs were obtained on both sides, but there was no ankle clonus at any time.

November 17, G. W. Holmes reported that pneumoventriculograms showed: The ventricles were rather large, and the right appeared to be somewhat larger than the left. No definite abnormalities in outline were visible.

November 21, cerebellar decompression was performed by Dr. J. S. Hodgson. Marked increase of intracranial pressure was found, but no pathological lesion was discovered.

The patient died November 25.

The necropsy was performed by Dr. Oscar Richardson. Examination of the head only was permitted. The brain presented marked flattening of convolutions, indicative of increased intracranial pressure. On the right side, section of the tentorium exposed a cyst-like tumor which rested between the basilar process and the brain-stem. The pedicle sprang from the region of the dura along the right side of the sella turcica and was continuous with the cyst wall. The tumor pressed upon the right crus and the upper part of

the pons, and down along the pons on the right and the superior surface of the right side of the cerebellum. The brain tissue in this region presented a pressure cavity. Otherwise the brain was not abnormal.

After hardening in formaldehyd the cyst was further examined. It was ovoid in shape and 5 by 4 cm. in size. Its contents were yellow and gelatinous, and contained numerous cells filled with fatty granules. No hooklets were



found. The pedicle of the cyst was about 1 cm. in diameter and was composed of cells of uniform character, with small oval or round nuclei and large granular protoplasmic bodies. Interspersed with these cells were bundles of glia and connective tissue fibers. No mitotic figures were seen. In the tumor no areas of degeneration were visible. The cyst wall was of almost uniform thickness, from 3 to 4 mm.; the outer capsule was smooth and covered with connective tissue; the inner wall was rough, but showed no transverse bands. Microscopically the wall of the cyst resembled the pedicle.

Summarizing, it may be said that this patient presented an unmistakable clinical picture of progressive brain tumor localized in the right cerebellar fossa. The cyst, which unfortunately was not found at the operation, is somewhat unusual. It did not at any point invade the brain, and certainly did not arise from the acoustic nerve. Its origin seems to have been in the meninges, and its obvious attachment was to the dura in the neighborhood of the sella turcica. In the absence of a complete postmortem examination it is not possible to state that we are not dealing with metastasis, but the evidence at hand suggests that it is a primary tumor of the meninges, pathologically benign in character.

EXAMINATION OF THE HEART AND AORTIC ARCH BY ROENTGEN RAYS IN NEUROSYPHILIS. DR. C. A. McDONALD.

Roentgenograms of the heart and aortic arch were taken in forty unselected cases of neurosyphilis at the out-patient department of the Massachusetts General Hospital. In eighteen of the forty cases the roentgenogram revealed specific aortic disease; in these eighteen, physical signs of cardiovascular disease were recorded as present in six cases and subjective symptoms of cardiovascular disease in four cases. Sixteen cases were interpreted as arteriosclerosis of the arch, and in them there was no record of signs or symptoms of cardiovascular disease. Two cases had other heart conditions, and in one of these there were physical signs of heart disease. One case showed no arch or cardiac pathology. In two cases no roentgenogram interpretations were given, and in one case the interpretation was given without cardiac measurements. In these last three cases there was no record of physical signs or symptoms of cardiovascular disease.

Summarizing: (1) This study of the records of forty cases of neurosyphilis reveals physical signs of cardiovascular disease in 15 per cent. and symptoms in 10 per cent. (2) The roentgen-ray examination revealed specific aortic disease in 45 per cent., and arteriosclerosis of the arch in 5 per cent. of the cases. (3) Therefore, in 90 per cent. of the forty cases cardiac disease was demonstrated by roentgen ray.

RESUMÉ OF SOCIAL SERVICE WORK IN THE NEUROLOGICAL CLINIC AT THE MASSACHUSETTS GENERAL HOSPITAL DURING 1922. CHARLINE F. BUCK.

The total number of patients in the neurologic clinic to whom social service has been rendered thus far in 1922 is 391. The aims have been threefold: (1) To assist the doctor by making treatment available to and effective for the patient. (2) To instruct students and volunteers in the processes of social work. (3) To contribute material for medical social research.

The methods have varied according to the types of medical case which have been grouped as follows: I. Toxic-organic, with 209 patients, including syphilitic 69, chorea 40, epilepsy 35, paralysis agitans, multiple sclerosis, etc., 65. In this group the efforts were especially directed toward making medical care possible and continuous, or toward arranging institutional care elsewhere if advisable. For instance, financial arrangements for antisiphilitic treatment; loans to the patient or the provision of state medicines; intensive supervision of the hygiene of the choreic; or change of employment for the epileptic. II. Hypophrenic, with forty-seven patients. Efforts here were centered on the investigation of the home care of the child, persuasion of the family

to accept institutional care, the securing of special class training in the public schools, and arrangements for special supervision of adolescents. III. Functional, with 135 patients. Efforts in this group were directed, first, toward understanding the patient's personality, habits, characteristics, and environment; then toward alleviation of the deficiencies and difficulties in his home, work, or play life, instruction in mental hygiene, and arrangement for convalescence or vacation when needed.

In addition to the usual difficulties belonging to Dr. E. E. Southard's five evils, a few of the resistances to be overcome were: (1) Lack of institutions for chronic cases. (2) Lack of convalescent or vacation resources for men. (3) Unwarranted prejudices against public institutions. (4) Lack of "light" jobs. (5) Lack of welfare, educational, and recreational resources in local communities. (6) Inability to secure the cooperation of social agencies in cases with unfavorable prognoses (feeble-minded adolescents).

Social workers have come to depend on definite medical recommendations as a basis for social work. These are sometimes difficult to get. This handicap has been overcome this year, in the group of psychoneurotic patients, by the establishment of an advisory committee of two staff-doctors who examine the case medically, hear the results of social investigation and action, and give definite advice for further action.

For convenience we have grouped the results with 344 patients (the other forty-seven cases are as yet too incomplete) into cases with:

- | | |
|---|------------|
| (1) Satisfactory adjustments | 151 or 43% |
| Group I | 94 or 51% |
| Group II | 22 or 55% |
| Group III | 36 or 31% |
| (2) Partially satisfactory adjustments..... | 135 or 36% |
| Group I | 64 or 31% |
| Group II | 16 or 40% |
| Group III | 56 or 49% |
| (3) Failures..... | 58 or 16% |
| Group I | 25 or 13% |
| Group II | 2 or .5% |
| Group III | 21 or 18% |

DISCUSSION

DR. E. W. TAYLOR: Certainly if these figures are correct this is a most satisfactory showing as compared with what was done fifteen years ago, when we did nothing socially for patients of this type.

DR. C. M. CAMPBELL: In a general hospital one can do a great deal more than at a psychopathic hospital. Social service in a department of a general hospital must have a very wide-reaching effect.

DR. J. B. AYER: These cases seem to demonstrate that the organic cases were better adjusted. What does Miss Buck think is the group in which social service does most for the patient? The first group would require half an hour's time; a functional case might require ten hours' time or more. In which group do you do the best job, and to what cases do you think you ought to devote most time?

MISS BUCK: To give a selfish answer, I prefer work with the functional cases. The statistics look discouraging, but what is accomplished with an individual is often most satisfactory.

DR. C. M. CAMPBELL: One of your three aims is scientific; to see what the study of these cases means for developing social service as a thing in itself; to which group does that apply?

MISS BUCK: I should say the functional group.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 22, 1922

N. S. YAWGER, M.D., *Vice President, in the Chair*

A CASE OF LIPODYSTROPHY. DR. C. A. PATTEN.

This case is presented because of its unusual endocrine interest and because it illustrates a disturbance of fat metabolism which ordinarily receives little attention. Positive facts in the history are as follows: A maternal uncle was a bedwetter until 9 years of age, this condition having been promptly relieved by circumcision. The father and mother are thin, high strung and nervous individuals. There are eight brothers and sisters, the oldest sister being deaf and dumb from birth. The second oldest brother had bladder difficulty until the age of 2, when acute retention of urine necessitated hospital treatment; following circumcision he had no further trouble. The third brother had nocturnal and diurnal incontinence until the age of four; his cure also followed circumcision. The second sister had nocturnal enuresis until the age of 18, and was relieved by an operation for hooded clitoris; she also had an enlarged thyroid gland. Another sister, aged 11½ years, occasionally wets the bed and has also a hooded clitoris.

The patient, aged 10, has been a bedwetter continuously until the past month. Two and a half months ago she was operated on for a hooded clitoris and was then treated in the Orthopedic Hospital for one month. During the past month she has wet the bed on but two occasions. She was brought to the orthopedic clinic by her mother who complained that besides enuresis, the child was fretful, peevish and high strung, and during the past year had been "growing all out of shape." During the past year she has become much less active on her feet and seemed to tire much more readily. This was in striking contrast to her former activity. Relative to her figure, the family noted that the child was very large around the buttocks, thighs and legs, but looked smaller in the trunk and arms. There was a gradual development of this condition until three months ago. Although not poorly nourished, there is a striking difference between the upper and lower halves of the body.

In the hospital she appeared anemic, had a capricious appetite and was a nail biter. The thyroid was distinctly palpable but there were no toxic symptoms. There was marked lordosis in the lumbar region and she had a protuberant abdomen, which is improving with appropriate exercises. The laboratory tests showed a normal sugar tolerance curve though a high percentage of sugar. Blood count: red cells, 4,870,000; white cells, 7,600; hemoglobin, 81 per cent.; polymorphonuclears 43, transitionals 1, lymphocytes 46, eosino-

phils 3, basophils 2. Fluid intake and output in twenty-four hours were 36 and 23 ounces, respectively. Urinalysis was negative; basal metabolism was within normal limits.

Lipodystrophy is much more frequent in women than in men, and usually develops around puberty. Many girls show a tendency to the excessive deposition of fat around the buttocks and lower extremities which is not present in the arms, trunk and head. Occasionally, disappearance of fat in the upper parts produces an appearance of over-deposit below. The over-deposit and waste take place in the subcutaneous tissues rather than in deeper structures, although in pseudomuscular hypertrophy there is, in addition to the subcutaneous, an increase of fat between the muscle fibers. The connection between the muscular dystrophies and lipodystrophy is well worthy of consideration, and I feel certain that future investigations will disclose a similar etiology.

The usual picture of pseudomuscular hypertrophy is that of atrophy of the muscles of the arms and trunk with apparent hypertrophy of the muscles in the lower extremities. In these cases there is no subcutaneous fat in the regions of atrophy, but a marked increase in the thighs and legs. The regions affected in the muscular dystrophies are the same as those involved in lipodystrophy. The difference in the sex incidence of the two conditions is noteworthy; the majority of muscular dystrophies occur in boys, and of the lipodystrophies in girls. Cases are recorded of wasting of fat in the face only, but, curiously enough, the hands and feet are never involved. The condition has not been observed in animals. Six or seven cases of lipodystrophy in males have been recorded, the most recent report having appeared in the *Revue Neurologique* of September, 1922, by Professor Christiansen of Copenhagen.

There seems to be some connection between gonadal and thyroid functions and the occurrence of lipodystrophy. As previously mentioned it develops very frequently around the age of puberty, but it is often observed at the time of the menopause in women and of the senium in men. The general descriptions and appearance of cases suggest thyroid influence. In my patient the thyroid is enlarged though puberty is not yet established; consequently one might, in accordance with Timme's theories of compensatory over-function, predict that there is a lack of ovarian secretion for which the thyroid gland is attempting to compensate.

DISCUSSION

DR. CHARLES S. POTTS: The relation of muscular dystrophy to endocrine gland disturbance is interesting. There has been work done along this line by McCrudden of Johns Hopkins and by Janney, Goodhart and Isaacson of New York. They found definite disturbance of carbohydrate metabolism, as shown by a hypoglycemia, which depended on impaired glycogenesis, the carbohydrates being changed into fat instead of glycogen. They found this due to disease of the adrenals and other endocrine glands. I gave adrenalin to one case at the Philadelphia Hospital, apparently with some improvement. In a case at the Polyclinic we made a metabolic study and found no change in the blood sugar.

DR. WILLIAM G. SPILLER: I am rather dubious about the diagnosis. Lipodystrophy is such an unusual disease that when a case is so abortive as this one the diagnosis must be questionable. The paper by Christiansen alluded to by Dr. Patten is the most recent. Christiansen mentions that in only one case of the six occurring in men, collected by Boissonnas, was there any deposit

of fat about the buttocks. I should find some difficulty in distinguishing between muscular dystrophy and lipodystrophy where the two conditions are associated in the same person. This child has no atrophy of the face. Facial atrophy is commonly found in lipodystrophy of men or women, but there is no case recorded in which only the face was atrophied. I wish Dr. Patten would make more clear to us that this is not a case of muscular dystrophy.

DR. C. A. PATTEN: In answer to Dr. Spiller, I feel that he is correct in suspecting the presence of a muscular dystrophy. Our first thought when we saw the child in the clinic was that we were dealing with a pseudo-hypertrophic muscular dystrophy; but we examined her repeatedly during a month in the ward and could not find any weakness or difficulty in muscular movements. The lordosis and relaxed abdominal walls together with large leg muscles certainly suggest a dystrophy, but under corrective exercises the abdominal tone has improved and the lordosis has been partly corrected. The latter was possibly a postural defect. It is difficult to conceive a systemic condition in which deposits of fat occur in localized regions, but the fact remains that they do develop.

In cases, other than lipodystrophy, known to have disturbances of function of the glands of internal secretion, similar phenomena are observed. In some women at the time of puberty or the menopause, this excessive fat deposit about the thighs and buttocks occurs. This suggests an endocrine relationship. I feel strongly that we shall find an endocrine basis for muscular dystrophies and lipodystrophy. In this case there is enlargement of the thyroid gland and the patient has as yet no apparent pubertal changes.

A case of pseudo-hypertrophic muscular dystrophy at the Polyclinic Hospital was able to get up from the floor without assistance, and to run considerably better after three months treatment with pituitary and thyroid substances. This is but one case, however, and improvement under encouragement must be considered.

As I understand it, the term lipodystrophy applies to a disturbance of fat metabolism. This disturbance may be an atrophy (or failure of deposit) of fatty tissue in one portion of the body, while in other regions there is an increase in the deposit. The regions involved in the atrophy and hypertrophy are the same as those of the muscular disturbances in some muscular dystrophies, and for this reason I infer that there is a possible etiologic relationship between the two conditions. It is significant from the gonadal standpoint that the lipodystrophies are much more frequently found in women, and the muscular dystrophies in men.

CHARCOT JOINT OF THE ELBOW IN A JEWISH WOMAN. DR. EUGENE LINDAUER.

A diagnosis of tabes was made in this Jewish woman twelve years ago; Eighteen months ago she had her first gastric crisis for which a hypodermic injection of morphin was given into the right arm. The next day the site of injection showed violent irritation; when the inflammation had subsided, the elbow joint remained enlarged and presented a typical Charcot joint. A spontaneous fracture developed in the joint later, and there was no subsequent union. The roentgen-ray picture of the elbow is typical of bones thus affected; similar bone disturbances are visible in the right knee joint and in the metatarsus of the right foot. No changes are visible in the wrist of the left side.

DISCUSSION

DR. SHERMAN F. GILPIN: This patient reminds me of a man, whom I saw a few months ago. He worked in a coal mine and believed himself in good health until he lifted a heavy object and hurt one elbow joint in the strain. A diagnosis of Charcot joint was made and confirmed by roentgen ray.

DR. GEORGE WILSON: A patient in Dr. Spiller's service at the Philadelphia Hospital at the present time has, among other trophic disturbances, a rupture of the left biceps muscle which developed after an injection of typhoid vaccine into the arm. This is interesting in relation to the history of the onset in Dr. Lindauer's case.

DR. J. HENDRIE LLOYD: In the roentgen-ray report the statement is made that the disease process in this joint is, in all probability, of syphilitic origin. I do not quite know what that means, but it suggests to me that Charcot joints may be due to the local action of spirochetes. I believe that someone may yet demonstrate the presence of spirochetes in these joints, as Noguchi demonstrated them in the brains of general paralytics. It is well worth a careful study. The theory that these joint disorganizations are due to "trophic" disorder is vague and unsatisfying. It is much more likely that this extensive destruction of the tissues of the joint is caused by a local agent which acts directly on these tissues.

DR. WILLIAM G. SPILLER: A year or two ago a man with an arthropathy of the elbow was in my service at the Philadelphia General Hospital. The fluid escaped spontaneously from the affected joint and amputation became necessary.

The patient in my service, to whom Dr. Wilson referred, has a rupture of his biceps muscle and an arthropathy of his lower jaw and of his thumb. The rupture of the biceps muscle, I think, may be explained by the tabetic incoordination between agonistic and antagonistic muscles, which prevents the proper degree of relaxation in the antagonistic muscles when the agonists contract. In this case the relaxation of the triceps muscle probably was insufficient to permit the contraction of the biceps muscle and rupture occurred in the latter. I have known the greater tuberosity of the humerus to be broken off by a sudden contraction of the flexor muscles when the opposing muscles did not relax speedily enough.

DR. EUGENE LINDAUER: In almost every case of this nature that I have seen the condition has been attributed to a real or imaginary injury. I recall a man on Dr. Potts' service some years ago who had a Charcot joint of the hip; a physician had applied a mustard plaster to the locality for the relief of pain; the next morning when the patient awoke he found a large mass in the hip and had difficulty in walking. He thought that the mustard plaster was the causative factor. The trophic nature of a Charcot joint is well known, but whether local or central I am not able to say. I believe that a local nidus of spirochetes might be found.

A CASE OF SENSORY DISTURBANCE LIMITED TO A PORTION OF
THE DISTRIBUTION OF THE ULNAR NERVE. DR. ALFRED GORDON.

A woman, aged 31, with a negative past history, began to have trouble with her hand at the age of 20. In the initial attack the fifth finger of the left hand became pale and cold, then bluish and swollen; the finger was incised because of the severe pain, and bloody serum was found. The wound healed

by first intention. Three months later she had a similar attack that involved the fifth and fourth fingers. Three years later the fifth, fourth and third fingers became involved. Five years later she had a similar involvement of the second, third, fourth, and fifth fingers. Two years ago, the patient noticed that when she lay upon her left shoulder and arm the upper extremity became numb and ached. In December, 1920, she had difficulty in grasping and holding objects with her left hand, and it was at this time that she noticed the loss of sensation. During the last six months her arm and forearm ached at times, especially after lifting objects of some weight above her head. She never noticed any blisters or sores upon her fingers.

Physical examination revealed a distinct and very pronounced anesthesia to touch, pain, and temperature on the hand and wrist, covering the area of distribution of the sensory fibers of the ulnar nerve over the palmar and dorsal surface and encroaching slightly on the median nerve distribution. Not only superficial sensation is abolished but there is also a total loss of all deep sensibility including the stereognostic sense. There is no atrophy of any of the muscles of the arm, forearm, or hand. Only a slight amount of weakness in the hand is present. The brachial plexus above and below the clavicle is painful to pressure. All tests, including the Wassermann, are negative. A roentgen-ray picture of the thorax shows a slightly exaggerated curvature of the first rib.

The case is interesting and unusual in view of the strict limitation of the sensory disorder and of its intensity, also in view of the absence of muscular disorder, notwithstanding the duration of the disease. Stookey in his recent book on peripheral nerves mentions disorders of the brachial plexus as a result of perfectly normal thoracic first ribs, in which fibrous bands have been found on the anterior end of the rib pressing directly upon the lower cord of the brachial plexus. The present case may be one of this group.

COMBINED SCLEROSIS OF THE SPINAL CORD OF THE ANEMIC TYPE WITHOUT BLOOD CHANGES. DR. N. W. WINKELMAN.

While there are patients that show the symptoms and signs of a postero-lateral sclerosis prior to the development of change in the blood, very few die without having some of the characteristic blood findings. Such a case I wish to relate. It concerns a man of 37 whom I first saw in March, 1921. He stated that he had been perfectly well until October, 1920, when he noticed numbness in the legs with difficulty in walking and a loss of sexual power. Gradually the disturbance in walking increased; he became uncertain in his gait and his feet felt extremely cold. When examined, the patient had a yellowish color; he was spastic and ataxic in his gait. The deep reflexes were exaggerated with bilateral clonus and Babinski sign. The sensory findings were rather characteristic: impairment of the sense of vibration and of position; tactile, temperature and pain sensibility normal. A diagnosis of postero-lateral sclerosis of the anemic type was made and he was taken to the University Hospital for further study. The red cell counts varied between 3,500,000 and 4,110,000 and the hemoglobin between 75 and 85 per cent. No alteration was seen in the blood smear. The Wassermann reactions of the blood and spinal fluid were negative. Gastric analysis showed absence of free hydrochloric acid. He left the hospital, and after many months was sent to the Philadelphia Hospital, when he was barely able to walk. At this time he showed symptoms indicative of a more or less complete lesion and was thought to have transverse myelitis. His blood did not show evidence of pernicious

anemia even four days prior to death, when the blood count was: red cells, 4,470,000; white cells, 10,300; hemoglobin, 80 per cent. He had developed bed sores and cystitis, which was followed by pyelitis from which he died. The necropsy revealed pyelonephritis; and histologically the spinal cord gave typical but rather advanced evidences of a so-called "combined sclerosis."

DISCUSSION

DR. WILLIAM G. SPILLER: Perhaps the most interesting feature in Dr. Winkelman's case is the excellent condition of the blood a few days before death at the last examination made. In most of the cases of anemic postero-lateral sclerosis in which the blood has been in good condition, a decided anemia has developed shortly before death. I have seen the blood picture fall suddenly a few weeks before the fatal termination. An explanation I would offer for this is that there is a certain reserve power in the blood-making organs and that the apparent good condition of the blood is deceptive. The blood-making organs use their reserve force and work under strain. They employ all the energy they have. Then comes a time when the reserve power is exhausted and the sudden fall in blood count and hemoglobin occurs, and is soon followed by death.

DR. CHARLES S. POTTS: Some of the members may recall a paper published by Putnam about 1891, in which he directed attention to the occurrence of cases of this kind due to causes other than pernicious anemia. I remember that he spoke of chronic diarrhea and malaria which of course might cause secondary anemia. I think Dr. McConnell showed a case to this Society which apparently followed a severe hemorrhage.

INJURY OF ONE PYRAMIDAL TRACT RESULTING FROM A KNITTING NEEDLE PUNCTURE WOUND OF THE CERVICAL REGION OF THE CORD. DR. J. C. YASKIN.

This case is of interest because it presents signs of a traumatic lesion localized in the right pyramidal tract of the cord about the level of the fifth or sixth cervical segments. The patient, 3 years of age, was in good health until Oct. 28, 1922, when he was accidentally struck on the right side of the neck with a knitting needle. He fell instantly and was dazed. Upon regaining consciousness, he was unable to sit up, and it was observed that he had lost power in his right arm and leg. When first examined at the Polyclinic Hospital two days later, he was somewhat dull and apathetic; he had a temperature of 100 and a pulse of 120; and was perspiring profusely on the right side of his head and face. The neck was rigid and the child cried out when an attempt was made to move it. There was a puncture wound of the skin about one-half inch above and one and one-half inches to the right of the sixth cervical vertebral prominence. There was no evidence of facial palsy but the right pupil and the right palpebral fissure were distinctly smaller than those on the opposite side. There was complete flaccid paralysis of the right arm and right leg. There was absence of all tendon reflexes on the right side, and of the cremasteric and abdominal reflexes on both sides. There was a well marked Babinski sign on both sides. As nearly as could be ascertained there was no sensory disturbance on the right side, and no failure of response to pain and temperature on the left. The patient was incontinent from the onset of the trouble. Roentgen-ray examination of the neck and skull was essentially negative; the eye grounds were normal; a lumbar puncture was deemed inadvisable on account of the condition of the patient.

The child did not show improvement until November 4, when he began to move his fingers and raise the right arm at the shoulder. About two weeks later there was evidence of tonicity on the paralyzed side and it was possible to obtain the biceps, triceps and patellar reflexes. In the latter part of November, the right side became definitely spastic, and about December 4, the Babinski sign had disappeared on the left side.

Physical examination now reveals: The left extremities are normal. The right extremities are spastic and paretic but the patient is able to use his arm and to walk with assistance. The biceps and triceps reflexes are present but are not exaggerated. There is a slight flexor contracture of the fingers of the right hand. The right abdominal and cremasteric reflexes are entirely absent. The ankle jerk is about normal. There is a well marked ankle clonus and Babinski sign on the right side. There are no tremors, atrophy, evidences of incoordination, nor of disturbances of sensation. The patient is still incontinent but, owing to his age, there is a question as to whether this condition is due to organic disease.

From the history, clinical course and observation of this case, it would appear that the needle had penetrated to the cord injuring only the right pyramidal tract. The absence of trophic disturbances, as well as the absence of root pains and disturbances of sensation, would lead to the conclusion that there was no involvement either of the gray matter, nor of any of the other tracts. The presence of the Babinski sign on the opposite side was probably due to a temporary edema and the ocular symptoms shortly after the injury were due to the involvement of the sympathetic system.

Book Reviews

BRAIN ABSCESS. Its Surgical Pathology and Operative Technic. By WELLS P. EAGLETON, M.D., Lt.-Colonel, M. R. C., Medical Director, Newark Eye and Ear Infirmary, Newark, N. J. Cloth. Price, \$7.00. Pp. 297, with 40 illustrations. New York: The Macmillan Company, 1922.

The author was possessed of considerable enthusiasm for his subject when he wrote this book, but enthusiasm is essential if one expects to present something worth while. Throughout the work, the author constantly tries to impress on the reader the great importance of detail; therefore, the most minute point in technic is carefully set forth and emphasized. This is an excellent feature, for such attention to detail is seldom found in works on general surgery, and in no field of surgery is detail more essential to success than in surgery of the brain.

While the author's classification of brain abscesses has some merit, it leads to much repetition of description, but perhaps he believed that only in that way could his views be sufficiently impressed on the reader. At times the text is somewhat disappointing, for instance: "In the surgical treatment of encapsulated abscess one of two principles is employed: 1. Attempted eradication of the suppuration. 2. Drainage." The advice is good, but one is left somewhat at sea when the advice is followed by the statement that "Drainage is generally inadequate—and may be the cause of secondary infection of the meninges, the development of the so-called secondary abscess and of hernia cerebri" and that "Complete evacuation, cleansing, and obliteration are impossible." Discordant views are found occasionally, such as "Metastatic abscess follows the occlusion of a cerebral vessel by an infected nidus circulating freely within the blood stream." "The vast majority of, if not all, metastatic brain abscesses are undoubtedly of venous origin—this view explains and is substantiated by the relative infrequency of brain abscess in ulcerative endocarditis." "It is a clinical fact of great importance that while suppurative diseases of the lungs, particularly bronchiectasis, frequently cause abscess of the brain, suppurative endocarditis rarely does so, although the size and directness of the arteries from the heart to the brain would lead one to expect abscess of the brain to be a frequent complication of suppurative endocarditis. Such, however, is not the case." Then follows an analysis of sixty-seven cases of endocarditic emboli from the left side of the heart. Metastatic abscesses occurred in the kidneys fifty-seven times, in the spleen thirty-nine times, and in the brain fifteen times. These figures show that more than 20 per cent. of metastatic abscesses due to emboli from the left side of the heart are really in the brain, and hardly prove the statement that all metastatic abscesses of the brain are of venous origin.

The author's attempt to explain the cause of the subnormal temperature so frequently seen in patients with abscess of the brain on an assumed analogy between the conditions found in patients suffering with abscess of the brain and those found in hibernating animals is certainly far fetched. Conclusions based on analogies are always precarious, but when the analogy involves only a single relation out of many, the conclusion can have no weight.

Notwithstanding these slight defects, the work as a whole, which is well worth while, contains much valuable matter, and the earnest and painstaking manner in which the author presents his subject will go far toward carrying conviction. An excellent bibliography accompanies the text, and there are three appendixes. Appendix No. 1 is "A Guide for Detailed Neurological Examination"; Appendix No. 2, "An Analysis of Pathological Conditions in Reported Autopsies of 125 Cases of Cerebellar Abscess"; Appendix No. 3, "An Analysis of 140 Reported Cases of Frontal Lobe Abscess."

OUTWITTING OUR NERVES. JOSEPHINE A. JACKSON and HELEN M. SALISBURY. Cloth. Price, \$2.50. Pp. 403. New York: The Century Co., 1921.

Singular as it may seem, this book has received almost unanimous praise, not only from the newspapers and lay journals, but also from the medical press and even from the orthodox psychoanalytic press. It is a popular presentation of Freudian principles, simply written and coherently constructed. From the standpoint of the thoroughgoing Freudian it is an exceedingly sugar-coated presentation. Only the elementary principles of Freudian theory and practice are discussed, and in this sense the announcement that it is a "comprehensive presentation" is inaccurate. In the last third of the book illustrations are cited which deviate from the Freudian conceptions espoused in the first part, and approximate more to the technic and methods of Dubois, Dejerine and Janet.

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TERATOID CYST OF THE HYPOPHYSIS *

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Among the cystic tumors of the brain is a small group of neoplasms which, through their relation to the pituitary body or through pressure on it and adjacent structures, give rise to signs of disturbance of pituitary function and thus acquire an especial clinical interest.

Such cysts have, in the past, been variously described as cholesteatomas, dermoids, epidermoids, and, occasionally, because of the occurrence of cartilage and bone in their walls, as teratomas.

The finding of cholesterin crystals in the interior of such cysts led Mueller¹ to name them cholesteatomas. Virchow,² however, later rejected this term on the ground that the cholesterin crystals are not constant, and, when present, are simply the product of a degenerative process. He advocated a return to the old term of Cruveilhier "Tumeurs Perlées" (Perlgeschwülste).

Various hypotheses concerning the origin of these tumors have been advanced. Virchow believed them to arise by methaplasia from the pial or dural connective tissue. This view was opposed by Remak (cited by Erdheim) who thought that they were a variety of dermoid. Bonorden³ described a cyst in the vicinity of the olfactory nerve which was lined by stratified squamous epithelium in which he found many spherical, concentrically arranged collections of squamous epithelial cells, forming structures not unlike epithelial pearls. Underneath this epithelial lining were groups of sebaceous cells arranged in lobules and supported by interstitial connective tissue. The tumor in his opinion corresponded in structure to the so-called cholesteatomas and he concluded that it was ectodermal in origin because of the

* From the department of pathology and the neurologic service, Mount Sinai Hospital, New York.

* Work carried out during the tenure of the Sachs Fellowship in Neuro-pathology.

* Read before the New York Neurological Society, Jan. 10, 1922.

1. Mueller, Johannes: Ueber den feineren Bau und die Formen der krankhaften Geschwülste, Ed. 1, 1838, p. 50.

2. Virchow, Rudolph: Ueber Perlgeschwülste (Cholesteatoma Mueller), Virchows Arch. f. path. Anat. 8:371, 1855.

3. Bonorden: Ueber ein meningeales Cholesteatom mit Haaren und Talgdrüsen, Beitr. z. path. Anat. u. z. allg. Path. 11:593, 1892.

presence of epidermal derivatives, such as sebaceous glands and squamous epithelium. He suggested as possible sites of origin of those tumors the point of closure of the primitive medullary tube, Rathke's pouch and then hypophyseal duct.

Beneke⁴ maintained that the so-called meningeal cholesteatomas arise from the endothelial lining of the meninges. He based his opinion on the fact that by the employment of a silver stain he was able to demonstrate a strong similarity in the outline of the cells lining the cyst cavity and that of endothelial cells.

Later, however, he⁵ revised this opinion, accepting the view that some of these tumors, particularly those which contain hair and sebaceous glands, have an epidermal derivation, whereas a smaller group are of endothelial origin. He then suggested the division of such cholesteatomatous cysts into three groups: (1) meningeal dermoids containing hair and sebaceous glands, resulting from embryonal displacement of the skin; (2) meningeal endothelial-pearl tumors, free from hair and sebaceous glands, derived from the endothelial lining of the meningeal trabeculae, and (3) meningeal epidermoids free from hair and sebaceous glands, derived from misplaced epidermoid epithelial cells.

Bostroem⁶ in 1897 undertook the study of the histologic character of cholesteatomas with a view to determining their nature and origin. He decided in favor of the epidermoid character of such neoplasms. He laid particular emphasis on the work of Wilms,⁷ who had shown that dermoid cysts of the testes and ovaries almost always contain derivatives of all three germinal layers, and can be considered as true embryomas, differing from dermoid cysts elsewhere. The latter are bigeminal in origin and contain only ectodermal and mesodermal components.

He found further support for this conception in the fact that, coincident with the formation of the primary and secondary brain vesicles, there is a histogenetic change taking place in the adjacent enveloping ectoderm and mesoderm and that these zones of constriction of the medullary tube are the most constant sites of such tumors. These locations, in his opinion, are most favorable for embryonal misplacement of epidermis which may later develop into cholesteatomas. When, however, such epidermal nests give rise to new growths, the

4. Beneke: Zur Frage der meningealen Cholesteatome, *Virchows Arch. f. path. Anat.* **142**: 1895.

5. Beneke: Zur Frage der meningealen Cholesteatomen und Berichtigung, *Virchows Arch. f. path. Anat.* **149**:95, 1897.

6. Bostroem: Ueber piaie Epidermoide, Dermoide und Lipome und durale Dermoide, *Zentralbl. f. allg. Path. u. path. Anat.* **8**:1, 1897.

7. Wilms: Die teratoiden Geschwülste des Hoden mit Einschluss der sogenannten Cystoide und Enchondrome, *Beitr. z. path. Anat. u. z. allg. Path.* **19**:233, 1896.

resultant cysts should be grouped with the epidermoids; but when they contain connective tissue elements of the skin as well, they should be considered as dermoids.

Further light was thrown on the character and origin of cholesteatomas by the exhaustive work of Erdheim.⁸ His attention was directed mainly to the cysts which are found at the base of the brain, in the vicinity of the hypophysis, and which present, most commonly, a direct connection with some portion of the pituitary body. Such tumors have been frequently described as infundibular cysts, cystosarcomas, carcinomas, and, occasionally, as papillomas. These cystic tumors bear a strong resemblance to the so-called cholesteatomas because of their squamous epithelial lining, the presence of cholesterol crystals and the appearance in the lining membrane of pearlike structures. Occasionally, bone and foreign body giant cells are found in their walls. These tumors are usually well circumscribed and rarely invade the adjacent brain tissue. It is always possible to demonstrate in such tumors some connection with the pituitary, most often with the infundibular process. Erdheim's previous discovery of epithelial cell remnants in the thyroid region, in connection with the subinvolted thyroglossal ducts, led him to suspect a parallel condition in the hypophysis. He first studied normal hypophyses and showed that in about 80 per cent. of normal glands, small numbers of squamous epithelial cells were to be found on the anterior surface of the anterior lobe. He traced these epithelial rests to the subinvolted craniopharyngeal duct of the hypophysis. He then concluded that all so-called cystic cholesteatomas occurring in connection with the pituitary are derived from the subinvolted remnants of the hypophyseal duct, and designated them as hypophyseal duct neoplasms. He separated these tumors from the epidermoids of Bostrom by pointing out some differences in the gross anatomic relationship and variations in the histologic structure. The epidermoids, being true derivatives of the skin, he believed to give constantly the keratohyalin reaction in their epithelial cells, as well as other evidence of cornification. Hair and sebaceous glands and intercellular bridges between the squamous epithelial cells are also found. In the hypophyseal duct tumors he failed to get the keratohyalin reaction, and did not find hair follicles or sebaceous glands.

Erdheim, while crediting Bonorden with the early suggestion that there existed a definite relationship between cholesteatomas and the primitive buccal ectoderm, noted Bonorden's failure to substantiate this idea by actual histologic observations. He also disagrees with Bonorden's interpretation of the buccal ectodermal origin of all meningeal cholesteatomas, since misplaced epidermis associated with

8. Erdheim, J.: Ueber Hypophysenganggeschwülste und Hirncholesteatome, Sitzungsab. d. k. Akad. d. Wissensch. **113**: 1904.

embryonic development of the hypophysis will only explain the origin of such cysts as are found in the vicinity of the hypophysis and will not explain the origin of cysts found elsewhere. He also asserted that up to that time no epidermoid cyst of the hypophysis had been described, and that this serves as further proof of a distinct origin of the cysts occurring in connection with the hypophysis. He admits that, though the latter are also derived from ectoderm, they are not epidermal in origin, and, for that reason, they are not to be regarded as cholesteatomas, which must show their epidermoid character by the presence of keratohyalin and by evidence of cornification.

D'Orsay Hecht⁹ reported a case which showed striking similarity in its histologic character, anatomic relationships, and in the resulting clinical manifestations, to the case here reported. The microscopic study by Herzog of the tumor of this case demonstrated it to be composed of a variety of tissues. The parenchyma was mainly made up of what was thought to be derivatives of the anterior lobe of the hypophysis. There were numerous cells, apparently similar in structure to the cells of the anterior lobe, and there were groups of cells which by their outline and their lamellated concentric arrangement simulated epithelial pearls. There were also small ducts lined by cuboidal epithelium, and filled with a colloidal substance. In addition, there was found osteoid tissue with typical bone corpuscles and haversian canals. These findings indicate, however, that only two primitive germinal layers have contributed to its formation. Thus the tumor cannot be grouped with the true teratomas.

Ehlers¹⁰ reported a case which he considered to be an epidermoid cyst of the hypophysis because of the presence of keratohyalin granules.

Lewis¹¹ recorded a tumor of the hypophysis, which he considered to have originated from the intermediate lobe of the hypophysis because, in addition to the squamous epithelial pearls, he found numerous cells characterized by clear refractile cytoplasm, similar to the cells found in the intermediate lobe of the normal hypophysis.

Jackson¹² described a cyst of the hypophysis and concluded that tumors derived from the embryonal tissue along the course of the craniopharyngeal duct have not received proper recognition. He emphasized that tumors belonging to the hypophyseal duct group are always found in the median line at the base of the brain, bounded in

9. Hecht, D'Orsay: A Teratoma of the Hypophysis, *J. A. M. A.* **52**:1001 (April 8) 1909.

10. Ehlers, H. W. E.: Ein Beitrag zur Kenntniss der Infundibularzysten des menschlichen Gehirns, *Virchows Arch. f. path. Anat.* **199**:542, 1910.

11. Lewis, D. D.: A Contribution to the Subject of Tumors of the Hypophysis, *J. A. M. A.* **55**:1007 (Sept. 17) 1910.

12. Jackson, Harry: Craniopharyngeal Duct Tumors, *J. A. M. A.* **66**:1082 (April 8) 1916.

front by the optic chiasm and posteriorly by the pons. The hypophysis is usually intact, though it may be pressed against the floor of the sella. There is always an intimate connection between the tumor and the infundibulum. The first five cranial nerves are frequently involved. The cysts usually contain serous, mucous or hemorrhagic fluid, in which cholesterol crystals are often found. The inner wall of the cyst often shows papillary excrescences. Embryonal connective tissue and cells with intercellular bridging are also found.

Kanavel and Jackson¹³ reported three cases of cysts of the hypophysis, and believe that such cysts are legitimate operative risks.

The contribution of Duffy¹⁴ is of particular interest. In one of the several cases studied, he found structures believed to be derived from the pars intermedia in the wall of the cyst. These were gland-like structures lined by cuboidal or columnar epithelium and filled with a colloid substance. In addition, he found, in the substance of the growth, foreign body giant cells, cholesterol crystals, epithelial pearls, mucous connective tissue, calcified vessels and giant cells enveloping dying epithelial pearls.

In another tumor, in addition to the squamous epithelium and pearls, Duffy reports the presence of cells, sebaceous in character, and intercellular bridging of the epithelium. He believes that lipid materials from the cells are the source of the cholesterol crystals and that the latter stimulate the formation of foreign body giant cells. True bone with haversian canals was also present. The lack of actual typical skeletal bone formation he believes militates against the origin of such bone from true bone anlage.

This survey indicates the tendency of the more recent investigators, beginning with Erdheim, to single out one type of cholesteatoid cyst at the base of the brain, which because of its anatomic relationship to the hypothalamic region and because of its histologic features is thought to be derived from the remnants of the hypophyseal duct, and for this reason is designated as a hypophyseal duct tumor. There is also a tendency to distinguish this type of neoplasm from the dermoids and epidermoids of Bostroem, since they often fail to show keratohyalin granules in the lining epithelium. The absence of hair follicles in such neoplasms serves as another point of differentiation from dermoids elsewhere in meninges of the brain.

The tumor herewith reported presents many of the features common to the cystic hypophyseal duct tumor, but it is also related to the meningeal dermoids of Bostroem, as it contains keratohyalin granules and hair germs.

13. Kanavel and Jackson: Cysts of the Hypophysis, *Surg., Gynec. & Obst.* **26**:61-70 (Jan.) 1918.

14. Duffy, W. C.: Hypophyseal Duct Tumors, *Ann. Surg.* **72**:537 (Nov.), 725 (Dec.) 1920.

The case is also believed to be of importance from a physiologic point of view, for it bears out some of the conclusions reached by Bailey and Bremer¹⁵ in their experimental work on the pathogenesis of diabetes insipidus. They were able to show that lesions in the postinfundibular portion of the hypothalamus, and not modification of the posterior lobe of the pituitary, are responsible for the more manifest symptoms of the disease, such as polydipsia, polyuria and cachexia. It will be seen that in the case here recorded the pituitary gland showed no changes of pathologic significance, while the floor of the third ventricle and with it the postinfundibular region of the hypothalamus were markedly distorted, if not completely destroyed.

REPORT OF CASE

History.—A girl, aged 6 years, who had had no previous illness and had been quite normal up to the time of the onset of symptoms, six months before admission to the hospital, became markedly constipated. Sleep became disturbed, she became restless, lost her appetite and experienced constant thirst, demanding water frequently. Several weeks later, on consulting a physician, the diagnosis of diabetes was made, and the child was treated accordingly. With the increase of thirst and increase of water intake, there was an increase in volume and frequency of urination. The child was losing strength, and would frequently complain of fatigue; she gave up playing, became irritable, and finally became confined to bed because of constant headaches and general weakness. A week before admission, it was noted that the child's mouth was drawn to one side, and her left shoulder drooped and twitched frequently. She was admitted to the hospital with the complaint of headache, fatigue, excessive thirst, enuresis, loss of appetite and weakness of the left shoulder.

Physical Examination.—The patient was fairly well nourished and somewhat undersized, with a profuse growth of lanugo hairs all over the body. There was ptosis of the right eyelid and external strabismus of the right eye. The left pupil was larger than the right. Both pupils reacted to light and during accommodation. There was left facial weakness and slight weakness of the left arm and hand. The latter was held in the hemiplegic position. There was slight weakness of the left leg. The deep reflexes were more active on the left side than on the right, though generally reduced. The Babinski reflex was doubtful, but gait and station were normal. The abdominal reflexes could not be elicited. The spinal fluid was negative. The diagnosis of neoplasm involving the posterior lobe of the pituitary and the right crus cerebri was made.

Necropsy Findings (Examination of the brain only was permitted).—The exposed dura was normal in thickness and without demonstrable epidural or subdural hemorrhages. The underlying pia-arachnoid was markedly edematous and of a purple hue. The convolutions of the brain were definitely flattened and compressed. On raising the frontal lobes a large purple, fluctuating mass presented itself at the base of the brain. It covered the optic chiasm, to which it was adherent (Fig. 1). Posteriorly, it filled up the entire interpeduncular space. Its bulging inferior and partially free surface was prolonged into a

15. Bailey, Percival, and Bremer, Frédéric: Experimental Diabetes Insipidus. Arch. Int. Med. 28:773 (Dec. 15) 1921.

funnel-shaped process, which seemed to be continuous with the pituitary body. The latter was extremely small in size, compressed, and was lodged in a shallow and eroded sella turcica. The base of the superior surface of this mass was firmly implanted in the substance of the brain. The optic chiasm and tracts were flattened.

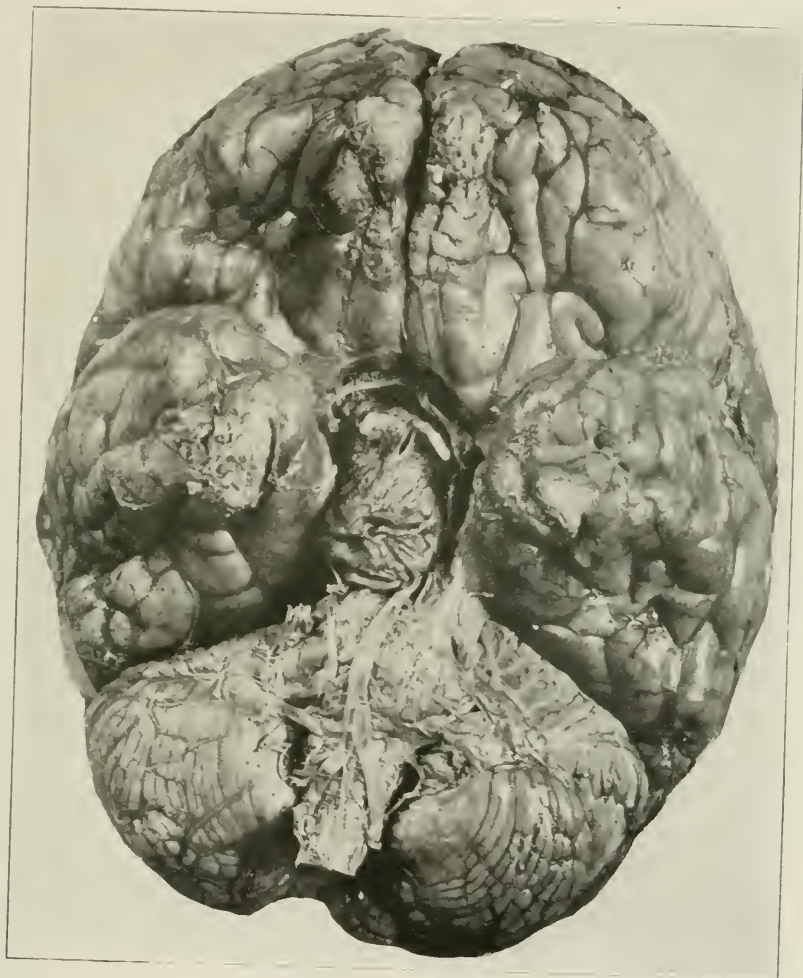


Fig. 1.—Ventral surface of the brain, with a cyst occupying the entire interpeduncular space.

The tuber cinereum and the mammillary bodies could not be identified since the floor of the third ventricle was stretched and flattened by the tumor mass, which almost obliterated the cavity of the third ventricle. The cerebral peduncles were displaced laterally and were reduced in size. This was particularly pronounced on the right side. During the process of detachment of the brain from the base of the skull, the neoplasm was punctured and a dark-brown, granular, semifluid mass escaped from the cavity. On opening the

latter more fully, it collapsed, and it was then noted that it was a rather thin walled cyst, lined by a corrugated membrane which was studded with numerous small, glistening elevations. Cholesterol crystals were demonstrated in the contents of the cyst. The cyst was 4 cm. in length and about 3.5 cm. in width. It had stretched markedly the vessels constituting the circle of Willis, particularly the posterior communicating and anterior cerebral vessels (Fig. 1).

The third nerve on the right side was compressed and flattened by the neoplasm. A horizontal, longitudinal section of the brain gave a still better

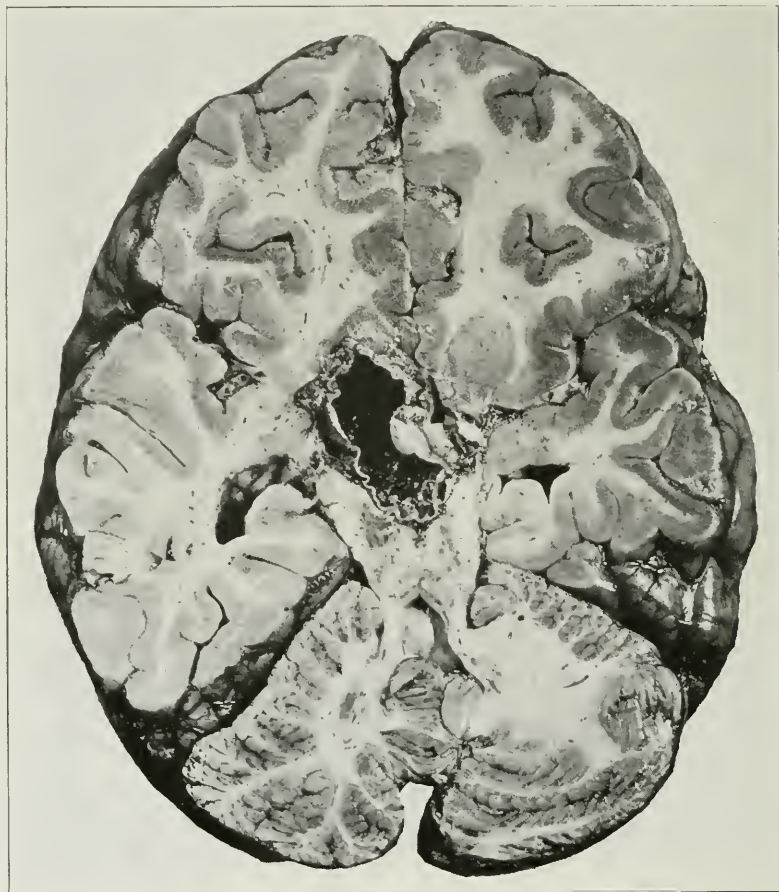


Fig. 2.—Section of the brain, showing the cavity of the cyst, the small cartilaginous mass projecting into the cavity, and the lateral displacement of the cerebral peduncles.

view of the gross structural changes that were brought about by the neoplasm (Fig 2). The cavity of the cyst was fully exposed and its inner wall was disclosed. The lining of the cyst gave the appearance of a mucous coat. At the left anterior portion of the cyst, the wall was thickened, giving rise to a tuberos elevation, cartilaginous in consistency, and somewhat translucent in appearance.

Microscopic Anatomy.—From the gross appearance of the neoplasm it was evident that little variation would be found in its makeup, except for the small cartilaginous nodule described above.¹⁶

The wall of the cyst was fairly uniform in thickness and showed also a more or less uniform histologic structure. It was composed of three layers. The innermost coat, a layer of stratified squamous epithelium, showed, at a few points, slight variation in the character and stage of development of its epithelium (Fig. 3). The middle layer consisted of loose connective tissue in which were embedded numerous glandular acini and many small ducts. The

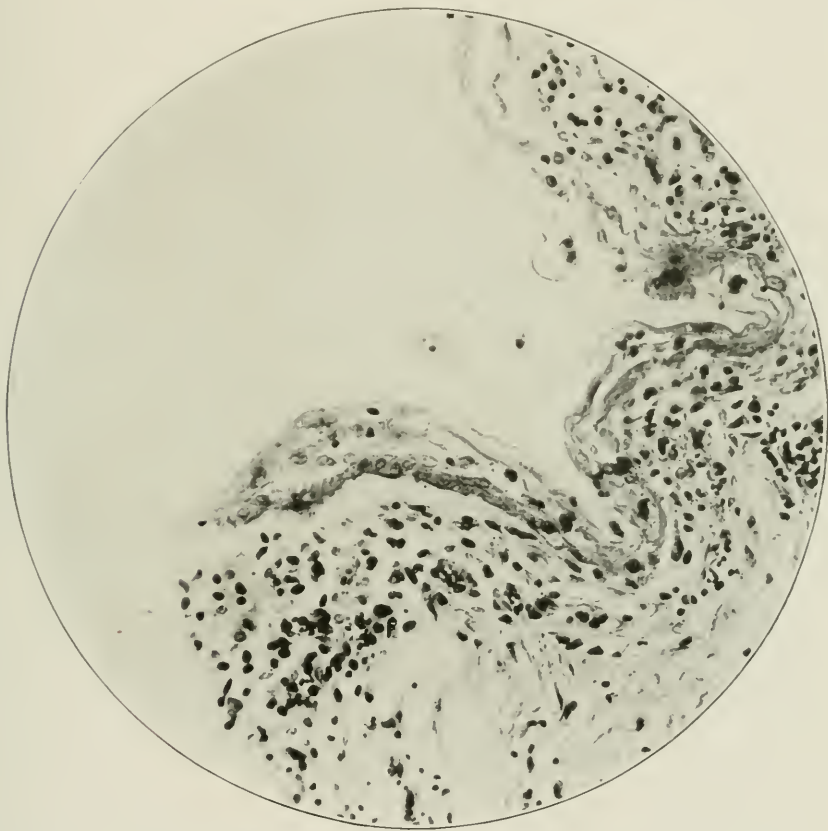


Fig. 3.—Photomicrograph of the inner lining of the cyst, showing the embryonal character of the stratified squamous epithelium; $\times 240$.

16. In order to make sure of this relative uniformity of the tumor structure, about twenty blocks were taken from various parts of the cyst, including several blocks of tissue from the cartilaginous mass. These were embedded in paraffin, cut in sections of various thickness and stained by such methods as hematoxylin-eosin, Heidenhain's iron-hematoxylin, van Gieson's, and Weigert's keratin stain. Sudan III and scharlach R were employed in staining frozen sections.

glands strongly resembled salivary glands and the ducts were lined by tall cuboidal epithelium. The lumen of these ducts was filled with a pink staining colloidal substance. The third and outermost layer was composed of a fairly thin stratum of dense fibrous connective tissues.

The squamous epithelium was of particular interest. This lining showed a strong resemblance to the dermis of an embryo (Fig. 3). It consists of three layers: the outer, corresponding to the epitrichium, the middle or intermediate layer and the lowermost or the stratum germinativum. Here and there

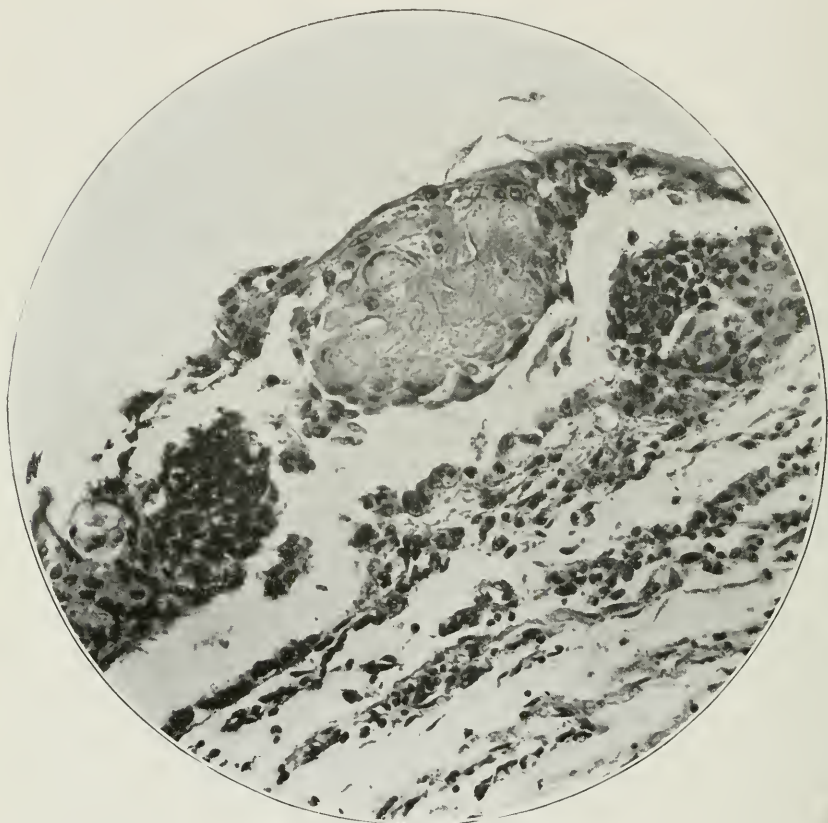


Fig. 4.—Photomicrograph showing well defined hair germs on either side of a group of degenerating squamous epithelial cells.

larger collections of cells composed mainly of the germinal layer gave the appearance of hair anlagen (Fig. 4). Another feature of interest was the accumulation of deeply staining epithelial cells arranged in concentric layers, giving rise to structures not unlike epithelial pearls (Fig. 5). The cells in the center of these pearls appeared to have undergone degeneration, while the peripheral cells have retained the structure of basal cell epithelium and show, when stained with iron-hematoxylin, the characteristic intercellular bridges;

when stained with Weigert's keratin method, a blue color was imparted to these cells, giving evidence of the presence of kerato-hyalin. These collections of epithelial cells were on the free surface of the cyst.

On studying the small cartilaginous mass in the wall of the cyst, there were found several types of embryonic tissues of mesodermal origin, such as embryonal cartilage, mucous connective tissue, young fibrous tissue, newly formed bone, with typical bone corpuscles, calcified trabeculae, endosteum and periosteum crowded with numerous osteoclasts, osteoblasts and osteophytes. Mar-

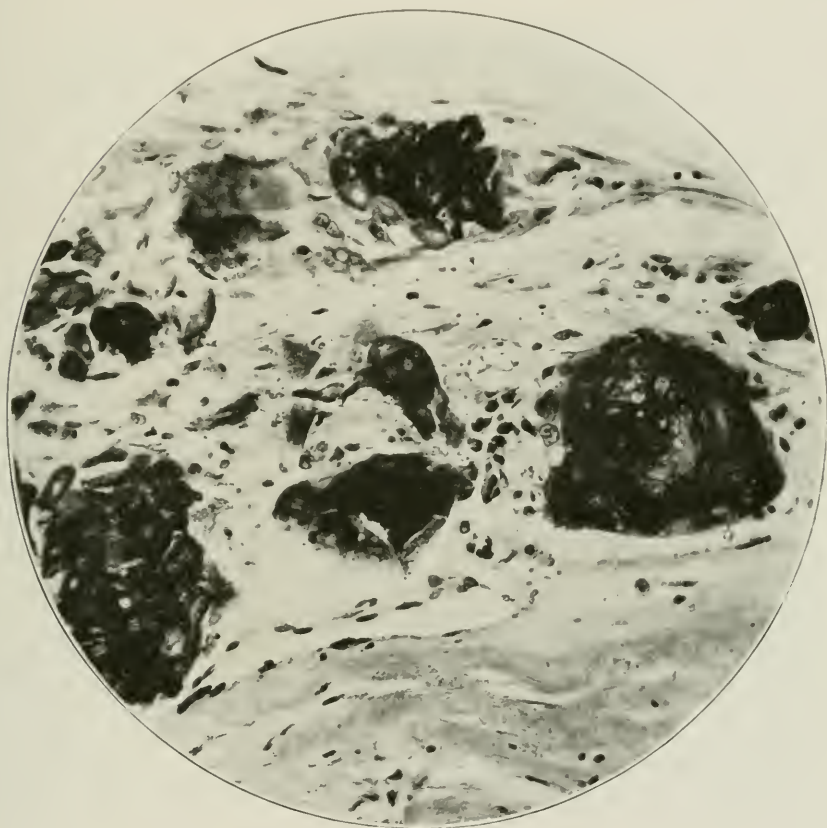


Fig. 5.—Photomicrograph showing the epithelial pearl-like structures in the innermost coat of the wall of the cyst; $\times 300$.

row cells filled with yellow granules, which gave a typical iron reaction, were also found. In addition to the structures noted, there was a collection of cells arranged in long cords and supported by a fine reticulum of connective tissue which, when stained by specific fat stains, were seen to be filled with large fat globules. These cells were first considered to be young embryonic fat cells, but because of the pyknotic character of the nuclei and the irregularity in the fat globules, they were finally thought to be similar to the cells found in sebaceous glands (Fig. 6).

Summary.—The tumor was a centrally situated, unilocular cyst, filled with dark brown, coarse, granular material. Its interior was lined by corrugated mucous membrane, on the surface of which were yellowish glistening papillary elevations. It bore a very intimate relationship to the hypophysis, being apparently attached to the infundibular process. Microscopically, it had a stratified squamous epithelial lining which was embryonal in character. Here and there on the surface of the epithelial lining, there was a heaping-up of cells which had a concentric lamellated arrangement, giving the appearance of epithelial

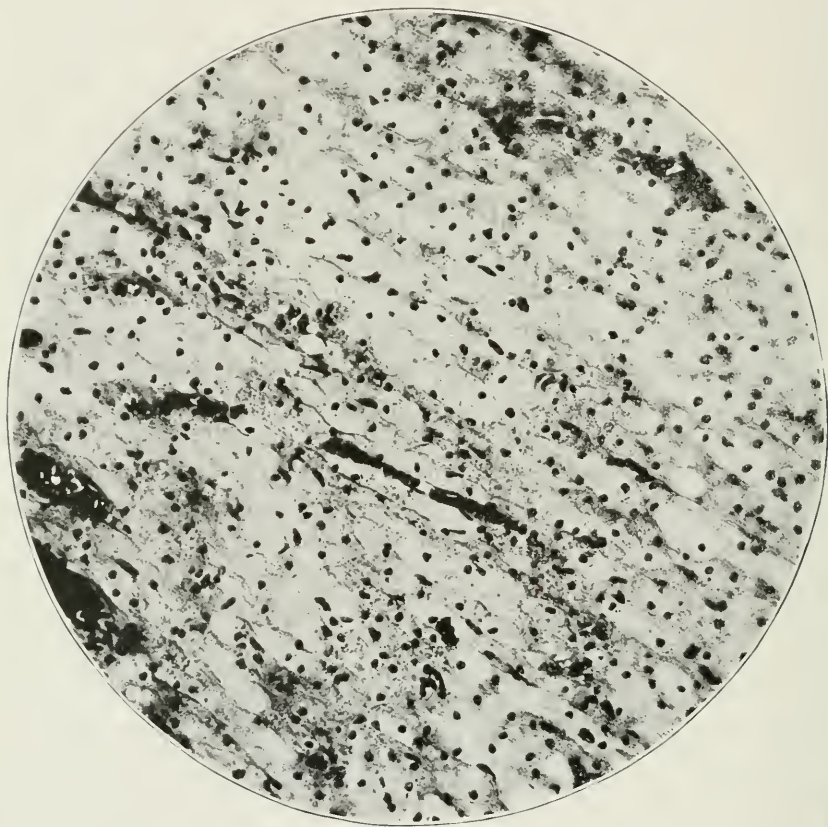


Fig. 6.—Photomicrograph of section through the small cartilaginous mass, showing cells similar to sebaceous gland cells, arranged in cords; $\times 240$.

pearls. The latter were in various stages of degeneration, showing central areas of calcification, and were frequently surrounded by foreign body giant cells. Beneath this epithelial lining, there was a connective tissue stroma, in which glandular tissue, of both salivary and sebaceous types were found.

The tumor also contained embryonal cartilage, dense fibrous tissue, embryonic mucous tissue, and bone, representing all the osteogenetic elements. Keratohyaline granules were demonstrated in squamous epithelial lining and

developing hair follicles were present in the form of hair germs. It is evident that this tumor presents in its structure all the elements of the ectodermal and mesodermal components of the primitive skin, to which other embryonal tissues of mesodermal origin (bone, cartilage, mucous connective tissue) are added. While it is quite possible that the calcified trabeculae and the osteogenetic elements are the result of metaplasia in a degenerated area, the hair follicles and sebaceous cells could not have had this origin. We are, therefore, dealing here with a growth which is bigerminial in character and not a true teratoma.

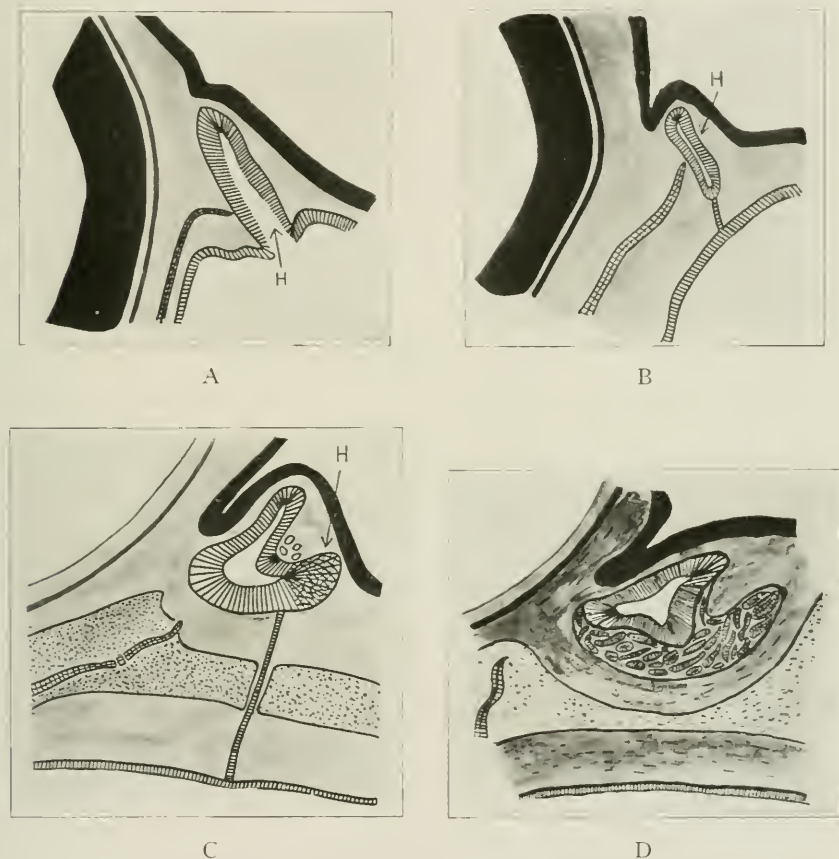


Fig. 7.—A series of diagrams illustrating the development of the hypophysis: *A*, invagination of the buccal ectoderm, giving rise to the so-called "Rathkes" pouch (*H*), communicating with the primitive oral cavity by the craniopharyngeal duct; *B*, Rathke's pouch which has assumed the form of a closed vesicle, with a solid cord of epithelium connecting the oral cavity with the hypophyseal vesicle; *C*, the anterior lobe of the hypophysis (*H*) beginning to differentiate in its anterior portion. The solid cord, representing the involuting craniopharyngeal duct, is still present, but is being surrounded by masses of cartilage and developing bone; *D*, complete involution of the craniopharyngeal duct. The anterior lobe of the hypophysis is beginning to gain its final glandular structure.

In comparing this neoplasm with those described by Erdheim, who believes the subinvolted craniopharyngeal duct to be the origin of such tumors, we find that there is striking similarity between the teratoid cyst here reported and his cysts derived from the hypophyseal duct. The only difference is that he failed to find hair follicles or keratohyalin granules in the epithelial lining, while these structures were found in the tumor here described. Erdheim lays emphasis on the keratin reaction, suggesting that the presence of this substance will differentiate between tumors derived from the subinvolted hypophyseal duct and the dermoids described by Bostroem. The hypophyseal duct tumors did not show hair follicles or keratohyalin granules while these were constantly present in the dermoids. This differentiation has already been disputed by Ehlers,¹⁰ who described a tumor similar to ours in which he was able to demonstrate keratohyalin material.

Ehlers came to the conclusion that in spite of the intimate relation of the tumor to the infundibulum and the possible origin of such a tumor from the primitive oral cavity, the presence of keratohyalin places this tumor with the dermoid cysts. He also suggested that the failure of Erdheim to demonstrate keratin can be accounted for by the method of fixation, as some of them had been kept for many years as museum specimens.

Bailey and Bremer¹⁵ have shown that the clinical symptoms of diabetes insipidus, such as polyuria and polydipsia, can be provoked with certainty by a lesion produced in the postinfundibular region of the hypothalamus and that the removal of the posterior lobe of the hypophysis does not lead to the permanent production of such symptoms. They conclude that there is no evidence supporting the older views which assert that a hypophyseal lesion is responsible for the symptoms of diabetes insipidus. The case here reported offers further support for the views expressed by the foregoing authors, for polydipsia and polyuria were striking symptoms late in the course of the disease and the anatomic findings showed that the pituitary gland was intact though somewhat compressed, but the hypothalamus was distinctly involved.

CONCLUSIONS

1. The histologic features of the tumor place it among autochthonous teratoid cysts.
2. Though the tumor probably had its origin from the lining of the primitive oral cavity, there is no histologic justification for placing it in a group distinct from other meningeal dermoids.
3. The occurrence of symptoms of diabetes insipidus in these hypophyseal duct tumors is probably referable to destruction in the hypothalamic region and not to destruction of the hypophysis itself.

CHARCOT-MARIE ATROPHY: THE REPORT OF A CASE WITH NECROPSY*

CHARLES S. POTTS, M.D., AND GEORGE WILSON, M.D.

PHILADELPHIA

Cases of Charcot-Marie atrophy with necropsy are sufficiently rare to warrant report. To date, six cases of this condition with the pathologic findings have been recorded; that reported by one of us (G. W.) is the only example from this country. The pathologic findings in the six cases have not been entirely uniform; all of them showed degeneration in the posterior columns, and in four of the six cases the peripheral nerves were diseased; in one slight degeneration was found in the peroneal nerves alone, and in one the nerve trunks were normal though the intramuscular filaments were altered. The anterior horn cells were greatly involved in one case, but the other five cases showed little alteration in the structure of these cells. Clark's columns were moderately affected in one case. The pyramidal tracts showed slight changes in two cases.

REPORT OF A CASE

Clinical History.—M. R., a single woman aged 51, was admitted to the Philadelphia General Hospital on July 9, 1914. At that time a note was made that a history could not be obtained because of the mental condition of the patient. In 1918, the history of M. R. which follows was obtained from her mother: The father died from unknown cause at the age of 38. The patient is one of five living children, aged respectively 60, 58, 57, 51 and 48 years. One child died in spasms when 18 months old and the mother had one miscarriage. No member of the family had had symptoms similar to those of the patient. She had the usual diseases of childhood, was bright, and received a common school education. As a child her legs were weak and she had to be helped up and down stairs by members of the family. She assisted with the housework but was never employed outside the home. Two years before going to the hospital she became bedridden and apparently suffered severe pain.

On July 26, 1918, one of us (C. S. P.) examined the patient and found: The woman was deficient mentally and unable to give a history of her illness. Vision appeared to be good and the cranial nerve functions were normal with the exception that a moderate degree of deafness was present. The pupils were contracted, irregular, and did not react to light, but reacted well in accommodation. The eyegrounds were normal. The patient was unable to walk or to feed herself. All movements of the arms could be made, though with diminished strength, but there was very little movement of the hands or fingers.

*From the Neurological Service and the Laboratory of the Philadelphia General Hospital and from the School of Medicine of the University of Pennsylvania.

*Reported at a meeting of the Philadelphia Neurological Society, Dec. 19, 1919.

Hyperextension was present at the metacarpophalangeal joint of all fingers except the index fingers; the second and third joints of all the fingers were flexed. The fingers, with the exception of the little and ring fingers of the left hand, could be passively straightened. The muscles of the shoulder girdle and arms were slightly atrophied; atrophy was much more intense below than above the elbows and fibrillary twitchings were present in the muscles of the forearms. The muscles of the back were well developed. Slight



Fig. 1.—Section of the second lumbar segment of the spinal cord.



Fig. 2.—Section of the cord from the mid-dorsal region.

power was present in the muscles of the legs, but was practically absent in those of the feet. The thighs were flexed on the abdomen and the legs on the thighs; the knees were ankylosed in a state of semi-flexion, and the feet were in the position of talipes cavus with slight varus. Atrophy and fibrillary twitchings were present in the muscles of the legs, especially in the peroneal and anterior tibial muscles. The patellar and achilles reflexes were absent and plantar stimulation produced flexion on both sides. Incontinence of urine was present, possibly in part due to the patient's mental condition, and in



Fig. 3.—Section of the first dorsal segment of the cord.



Fig. 4.—Low power drawing of the ulnar nerve.

part to lack of team work between the patient and the attendants. She apparently perceived painful stimuli but other forms of sensation could not be tested owing to lack of cooperation. None of the muscles of the legs, forearms and hands reacted to the faradic current. The Wassermann reaction with both blood and spinal fluid was negative, and no cells were found in the fluid. The patient died May 24, 1920, as the result of broncho-pneumonia.

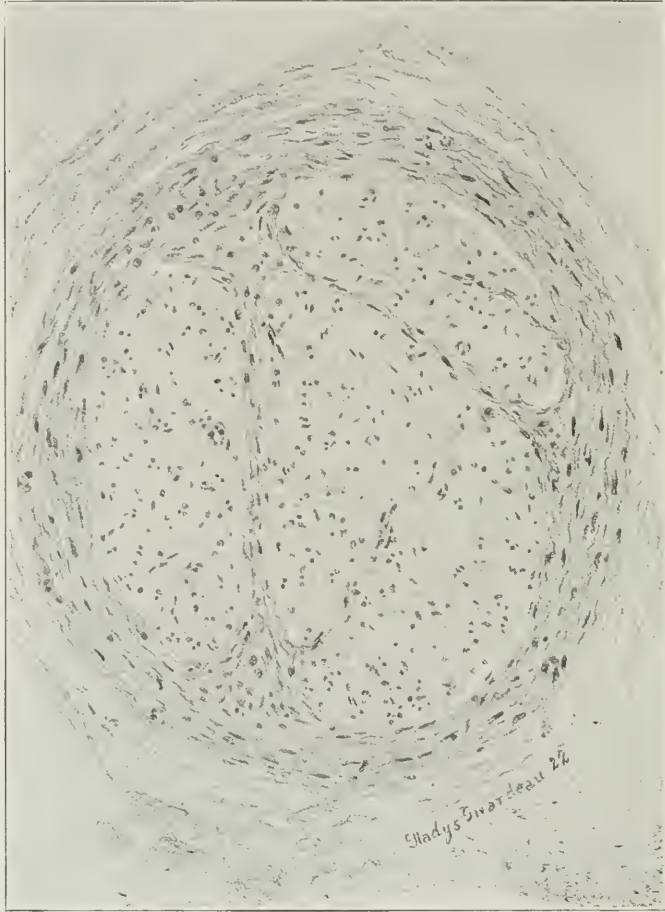


Fig. 5.—High power drawing of the ulnar nerve.

Pathologic Study of the Nervous System.—The spinal cord and the posterior and anterior roots appeared small on gross examination. The meninges, the cells of Clark's columns, and the pyramidal tracts were normal. The anterior horn cells in the lumbar, sacral and cervical regions were diminished in size and number; the nuclei were eccentric, the cell bodies stained poorly and consisted chiefly of a granular material. The changes in the anterior horn cells were most marked in those segments concerned with the muscles of the feet, legs, hands and forearms. In the upper cervical and upper lumbar regions the

cells of the anterior horns were altered but not so severely as those already described. The posterior columns were moderately degenerated throughout all regions of the cord. The degeneration was usually confined to the region about the posterior septum, but in the cervical levels, Burdach's columns were also involved. Lissauer's zones were greatly sclerosed in the sacral, lumbar and cervical regions. The cerebrum showed no changes of note.

The plantar, peroneal, median and ulnar nerves were degenerated, the plantar and peroneal nerves being intensely involved. The plantar nerves contained few healthy fibers; the median and ulnar showed a reduction in the number of healthy fibers but not to the severe degree observed in the plantar and peroneal nerves. The perineurium was greatly thickened in all the nerves examined; the epineurium showed slight hypertrophy, and the endoneurium was not appreciably altered.

Tissue removed from the plantar surface of the foot, and from the thenar and hypothenar eminences showed practically no muscle tissue at all; fat and connective tissue were the only structures present.

COMMENT

The pathologic changes in our case therefore corresponded with the two findings most commonly reported in cases of Charcot-Marie atrophy in which necropsies have been recorded: degeneration of the peripheral nerves and of the posterior columns of the cord. The cells of the anterior horns were more involved in this case than in the one reported by Wilson in 1918.

Clinically, our case showed the presence of mental changes and Argyll-Robertson pupils; these have occasionally been observed in other cases of peroneal muscular atrophy. Indeed, a number of cases have been reported with such atypical manifestations as increased reflexes, Babinski's sign, optic atrophy, ocular palsies and bulbar symptoms. The occasional appearance of these unusual symptoms in Charcot-Marie atrophy may be explained on the ground that hereditary diseases of the nervous system are, at times, blended together; hence atypical findings are to be expected.

RECENT DEVELOPMENTS IN ELECTRODIAGNOSIS*

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It has long been known that in various pathologic conditions the electrical excitability of nerves and the muscles innervated by them is greatly altered. Methods have also been devised by means of which variations in the excitability of these organs may be estimated. They have not, however, come into very general use in the United States.

One of the reasons for the relative neglect of electricity as a diagnostic aid in this country has undoubtedly been the recent astonishing development of the semiology of the nervous system, which has given us a vast number of objective signs and symptoms of derangement of function in various locations. Another reason was the lack of accuracy of the electrical measurements. With the faradic current, the distance of separation of the coils of the inductor may give a fair estimate of the electromotive force developed, but no measure of the time is possible, and the exciting force varies greatly with the physical characteristics of the apparatus and the resistance of the tissues. Even with the simple galvanic current, the size of the electrodes, their position, etc., cause great variation in the findings.

As a result of the inaccuracy of the electrical determinations and the greater ease with which equally valuable information could be obtained by simple clinical examination, there was a tendency for clinicians to ignore electrodiagnosis. Recently, however, particularly in France, great progress has been made in the development of accurate methods, so that today one may determine the functional condition of a nerve or muscle in terms of its electrical excitability with a mathematical precision which objective clinical examination cannot hope to attain. With the exception of the brilliant researches of Keith Lucas,¹ this progress has been made almost solely by the electrophysiologists of France.

The sole contribution to the subject, at least from the clinical point of view, in this country is the recent and interesting work of Sachs and Malone.² Certain views supplementary to those exposed in their

* Reported at a meeting of the Boston Society of Psychiatry and Neurology, Oct. 19, 1922.

1. Lucas, Keith: J. Physiol. **37**:460, 1908.

2. Sachs, Ernest, and Malone, Julian Y.: A More Accurate Clinical Method of Diagnosis of Peripheral Nerve Lesions and of Determining the Early Recovery of a Degenerated Nerve With Report of Cases and Experimental Data, Arch. Neurol. & Psychiat. **7**:58 (Jan.) 1922.

work have seemed to me to need further consideration, particularly in view of recent French work on the subject. In their article, the condenser methods are dismissed as inaccurate. In this view they followed Adrian, who seems to have based his objections mainly on the work of Laugier. Since then, the condenser method has been greatly developed; and the new system found to give accurate results by Laugier³ himself. It is for this reason that I have thought it interesting to bring these recent developments to the attention of the English reading public. In order to understand the discussion which follows, and to appreciate the merits of the different methods to be described, it is necessary to keep in mind certain fundamental conceptions of current electricity.

ELECTRICAL WAVES USED IN ELECTRODIAGNOSIS

The simplest of all conditions in electrodiagnosis is obtained by the use of the galvanic current. One has to do with the current of constant intensity furnished by a battery of accumulators, which can be closed or opened by means of a hand key. At the moment of closing the circuit the current is established instantaneously, and the intensity rises instantaneously from O to the value determined by Ohm's law $I = \frac{V}{R}$ in which I represents the intensity of the current in amperes, V the difference in potential in volts, and R the resistance in ohms. The current then passes with a constant intensity until the circuit is opened. At the moment of opening, the intensity falls instantaneously to O . The time between the closing and opening of the circuit, during which the current passes, is spoken of as the time, and the portion of current corresponding is known as an electrical wave.

Such a wave may be represented graphically by a rectangle. The quantity of electricity in such a wave is easily calculated, the quantity being the product of the intensity by the time of passage, $q = it$. If in addition to the intensity we know the voltage V under which the current has passed, we can easily calculate the energy furnished, W , by the wave of electricity, the energy being equal to the voltage multiplied by the quantity (when the current is of constant intensity): $W = QV$. If we substitute for Q its equal IT and for V its equal RI , we may write $W = RI \times IT = RI^2T$.

Another type of electrical waves is much used in electrodiagnosis; that furnished by discharge of condensers. The condenser furnishes a wave which attains its maximum intensity instantaneously at the moment of discharge and falls away rapidly to O . It is much more difficult to calculate the quantity of electricity and the energy given by these waves but by means of the integral and differential calculus it may be done,

since each has a known logarithmic formula: $I = \frac{V}{R} e^{-\frac{t}{CR}}$.

3. Bourguignon and Laugier: *Compt. rend. Soc. de biol.*, March 5, 1921.

LAWS OF EXCITABILITY

Between 1845 and 1849, Dubois-Reymond,⁴ starting from the fact that the closing or opening of a current furnished by an induction apparatus or by a battery of accumulators caused excitation of a nerve included in the circuit, as evidenced by the contraction of the muscle which it innervated, established the famous laws of excitability which bear his name. Without going into their development, since they are familiar to every one, we may enumerate them as follows: 1. The excitation is produced solely by the variation in intensity of the current and not by the absolute value of the intensity. 2. The variation in intensity is the more effective, the more rapidly it is produced. 3. The time of passage of the current and consequently the quantity of electricity and the energy are of no importance in the process of excitation.

The application of these laws to the clinic is the classical search for the minimum intensity which will just give a contraction of the muscle in question when the galvanic current is closed with a hand key, the so-called threshold of excitability.

The laws of Dubois-Reymond have been confirmed for the times of current which he had at his command, but it will be noted that he was limited to the rapidity with which he could close and open the circuit with the hand. In 1864, Fick showed that for still shorter electrical waves, the time factor was of great importance in the process of excitation. Because of technical objections to his apparatus, his results were not generally accepted until Hoorweg⁵ in 1897, by the use of condenser discharges, finally proved that for waves of very short duration, the law of Dubois-Reymond was not valid.

LAW OF WEISS

It was not, however, until 1901 that Weiss⁶ determined accurately the rôle which the time of passage of the current plays in the process of excitation. He used a constant current, closed and opened by means of a pistol ball. The pistol was so arranged that, when fired, the ball would cut two wires; the cutting of the first wire closed the current, the cutting of the second opened it. The duration of passage of the current was easily calculated from the distance which separated the wires and the known speed of the ball. By means of this arrangement, he showed that the intensity which gives the threshold of excitation diminishes when one increases the duration of passage of the current, until a certain minimum of intensity is reached, when the threshold no longer changes no matter how much one increases the duration.

4. Dubois-Reymond: *Untersuch. über thier. Electr.* **1**:258, 1848.

5. Hoorweg: *Arch. de physiol.*, 1897, p. 269.

6. Weiss: *Arch. italiennes de biol.* **35**:413, 1901.

In representing these results graphically, the times as abscissae and the intensities giving the threshold as ordinates, Weiss established a curve of hyperbolic form (Fig. 1); from a certain duration of current the intensity no longer varies, the hyperbola therefore continues as a straight line. It was this straight section of the hyperbola with which Dubois-Reymond was experimenting. To this hyperbola of intensity corresponds a rectilinear of quantity. The relations of the intensity and quantity of electricity with the time of passage of current constitute

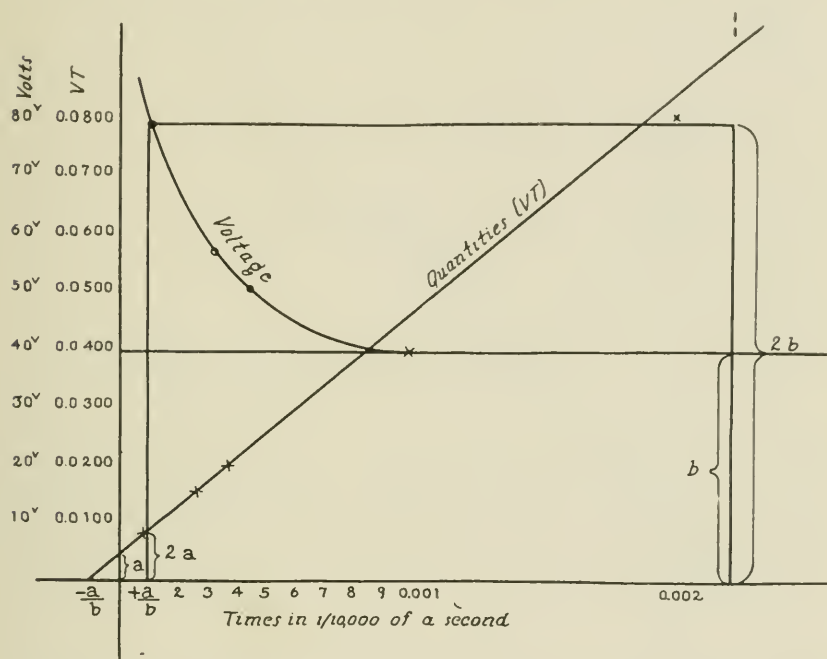


Fig. 1.—Law of Weiss. Right biceps (motor point). $a = 0.0035$. $b = 39.7$

$$\tau = \frac{a}{b} = 0.00009 \text{ (after Bourguignon).}$$

the law of Weiss, which is general for all tissues excitable by the electric current. These relations may be expressed by the following equations:

$$q = it = a + bt,$$

$$i = \frac{a}{t} + b,$$

in which t represents the time of passage of the current, i the intensity which has given the threshold with the time t , and a and b two constants, of which a is a constant of quantity in coulombs (the quantity which would be sufficient if the wave were infinitely short) and b a constant of intensity in ampères (the minimum of intensity after which the time no longer enters into consideration).

The constant b is familiar and easily determined. It is the galvanic threshold, called by Lapique the rheobase. The constant a is not directly measurable since it is impossible to employ a current of infinite shortness. It may be obtained by projecting the rectilinear of quantity across the line of infinite shortness of current (Fig. 1, a), or by calculation.

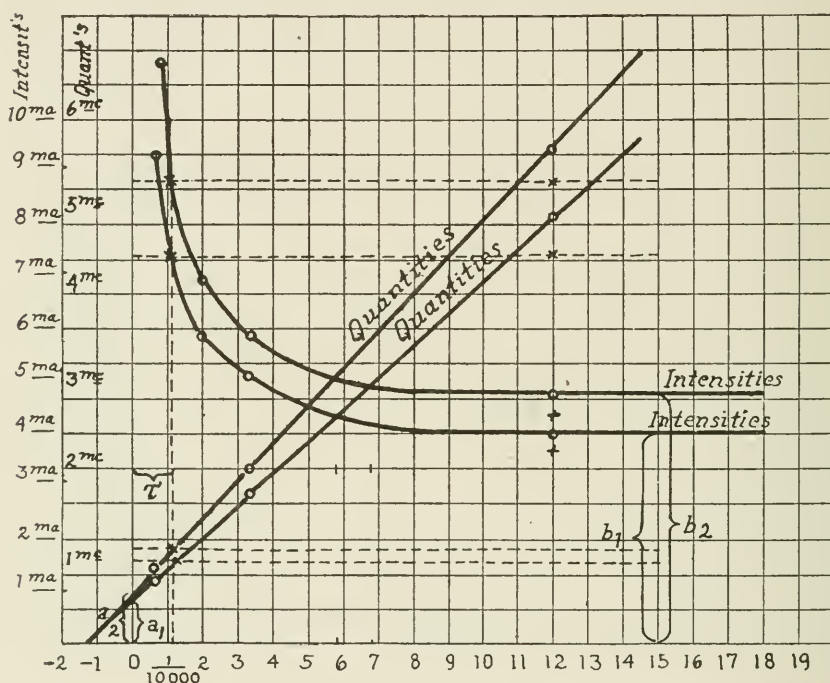


Fig. 2.—Two laws of Weiss obtained from the same muscle with two different positions of the active electrode—left supinator longus; man. Time in 0.0001 of a second. X, points determined experimentally. O, points calculated.

$a_1 = 0,^{mc} 444$. $a_2 = 0,^{mc} 527$. $b_1 = 3,^{ma} 7$. $b_2 = 4,^{ma} 39$. $\tau = \frac{a_1}{b_1} = \frac{a_2}{b_2} = 0,^s 00012$ (after Bourguignon).

The galvanic threshold b is therefore not sufficient to characterize the excitability of a tissue. Weiss also showed that while the value of a and b changed with the conditions of the experiment, the ratio $\frac{a}{b}$ of the two constants was invariable, and depended only on the excitability of the tissue examined. He, therefore, suggested that it was in the measure of this relation $\frac{a}{b}$ that one must seek for the characteristic of excitability of nerves and muscles, diseased and well. This relation $\frac{a}{b}$ was named by Lapique chronaxie, by which it is generally known. Being the product of a quantity by an intensity it is necessarily time in

seconds. Attempts have been made to express chronaxie in terms more accurate than those given in the formula of Weiss, but no one has succeeded in giving an equation at once so simple and so fruitful of results.

Weiss used rectangular waves in his determinations of chronaxie, but any type of electrical wave may be used, for example, the waves produced by condenser discharges. The results, however, will be in terms of the capacity of the condenser instead of time in seconds, and the calculation involved is complicated.

When one uses the current produced by the discharge of condensers, the quantity effective in producing the contraction is not the entire quantity of the discharge, for the tail of the discharge is inactive from the moment when the intensity has fallen below the rheobase. The quantity active Q , which corresponds to the formula $Q = a + bt$ is the quantity exerting its influence in the time between the initial moment of discharge (the potential at this moment may be designated V_o) and the moment when the voltage has fallen to the rheobasic voltage (V_{rh}). By having recourse to the logarithmic formula of the condenser discharge, Cluzet⁷ has shown that this time may be expressed $t = RC \log_e \frac{V_o}{V_{rh}}$. By substituting in the formula of Weiss:

$$Q \text{ or } C(V_o - V_{rh}) = a + bRC \log_e \frac{V_o}{V_{rh}},$$

in which C is the capacity of the condenser in farads, V_o the initial voltage of the discharge in volts, V_{rh} the rheobasic voltage in volts and R the resistance of the circuit in ohms.

From this may be derived the value of $\frac{a}{b}$ in terms all measurable:

$$\frac{a}{b} = RC \left[\frac{V_{rh}}{V_o} - \log_e \frac{V_{rh}}{V_o} - 1 \right]$$

in which R is the measured resistance of the circuit, C is the capacity of the condenser known in advance, V_o is the voltage of charge measured by a voltmeter and V_{rh} the rheobasic voltage measured by the same voltmeter using a constant current. If, now, we assign to V_o a value always the same, say the double of V_{rh} , the formula is simplified greatly and becomes:

$$\frac{a}{b} = RC (2 - \log_e 2 - 1) = 0.37 RC.$$

Lapicque⁸ determined experimentally what must be the capacity of a condenser capable of just causing an excitation, with a voltage double the rheobasic voltage, of a muscle with a given chronaxie. He found that the value of the chronaxie could be related to the capacity of the

7. Cluzet: Compt. rend. Acad. d. Sc. **137**:670, 1903.

8. Lapicque: Compt. rend. Soc. de biol., May 7, 1910.

condenser by the formula $\frac{a}{b} = 0.37 RC$. The formula is usually written $\tau = 0.37 RC$, in which τ is the chronaxie and C the capacity corresponding. The variation of the experimental result from that calculated mathematically is sufficiently small to be within the realm of experimental error.

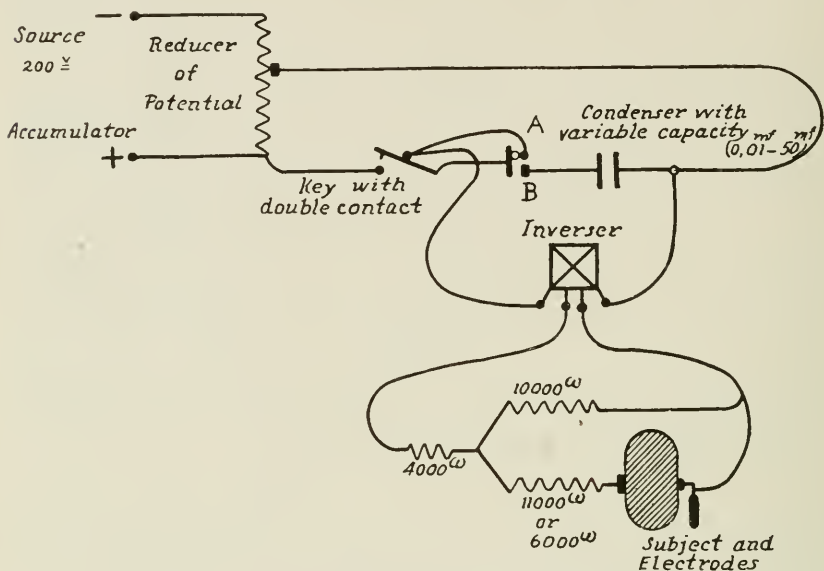


Fig. 3.—Scheme of connections for the determination of the chronaxie on the human subject, by means of the discharge of condensers, after eliminating the variations of the resistance of the subject (after Bourguignon). The connections are so made that with the commutator in the position *A* as represented in the figure, one opens and closes with the key the continuous current; in the position *B*, one charges and discharges the condenser.

THE CHRONAXIE

By a systematic study, Lapicque has shown that the relation $\frac{a}{b}$ is the only constant characteristic of any nerve or muscle, varying in a given tissue only with the temperature. When one makes a series of determinations on a given nerve or muscle, changing each time the experimental conditions, one finds as many hyperbolas of intensity and rectilinears of quantity as there are changes in experimental conditions, but all the hyperbolas are parallel and all the rectilinears converge at the same point: they converge at the point where they cut the axis of abscissae when they are prolonged to the negative side of the axis of ordinates (Fig. 2). There is, therefore, a length of abscissa, that is to say, a time of passage of current, which is constant for a given organ; it is this time which characterizes its excitability and has been called by Lapicque the chronaxie.

Now this point of convergence is exactly where $q=0$. Since $q=a+bt$, a simple calculation shows that when $q=0$, $t=-\frac{a}{b}$ we search, on the positive side, the intensity which gives the threshold, when the time of passage is equal to $\frac{a}{b}$, that is to say to the chronaxie, it is easy to see that it is when the intensity is equal to $2b$. For, if in the formula of Weiss we take $t=\frac{a}{b}$, the derivative formula $i=\frac{a}{t}+b$ becomes:

$$i = \frac{a}{\frac{a}{b}} + b.$$

Now $\frac{a}{\frac{a}{b}} = \frac{ab}{a} = b$; therefore $i = b + b = 2b$.

Lapicque⁹ has shown also that, having given the constant b (galvanic threshold) the name of rheobase or fundamental threshold, we may give an empiric definition of the rheobase and the chronaxie independent of all theoretic considerations of the law of excitation:

The rheobase is the intensity necessary to obtain the threshold of excitation on closing a prolonged constant current (the classic galvanic threshold).

The chronaxie is the time of passage of current necessary to obtain the threshold with an intensity double that of the rheobase.

In order, therefore, to determine the chronaxie, it suffices to find the galvanic threshold with the closure of a key, then double the voltage corresponding and determine the time of passage of current necessary to obtain the threshold with a voltage double the rheobasic voltage. For this purpose it is necessary to have at one's disposal a method of obtaining wave lengths in the neighborhood of 0.00001 of a second.

METHODS OF OBTAINING SHORT INTERVALS OF TIME

In order to obtain the minute intervals of current necessary in determining the chronaxie, one may use the pistol of Weiss. This method gives rectangular waves whose length can be measured very accurately. It has been described in the foregoing.

Keith Lucas¹ devised an instrument for this purpose generally known as the Lucas pendulum. It consists of a heavy metal pendulum mounted on a metal wheel in the manner of a hand on a clock. On the rim of the wheel are two keys, the first of which closes the circuit and the second opens it. The pendulum in falling strikes one key after the other. By varying the distance separating the keys any length of time of current may be obtained which is desired, and the time calculated from the known rapidity of falling bodies.

9. Lapicque: Compt. rend. Soc. de biol., July 24, 1909.

A similar instrument, with the wheel mounted horizontally and the hand driven by a falling weight, was devised by Lapique¹⁰ and called a chronaximètre.

Ströhl¹¹ has described an apparatus which he calls an égersimètre which replaces most advantageously the pistol of Weiss. A movable weight, moving without friction around a central column, throws the necessary keys, in falling from a height of 18 cm. The interval between the keys is regulated by displacing the movable one on a micrometer scale.

One may also use condenser discharges for the purpose. The time of discharge of a condenser being proportional exclusively to its capacity, it suffices to obtain the capacity which will give the threshold with a voltage double that which has given the threshold with the galvanic current. That capacity may be transformed into time in seconds, that is to say the chronaxie, by means of the formula developed in the foregoing $\tau = RC \tau 0.37$.

APPLICATION TO THE CLINIC

The foregoing work was done, for the most part, on the isolated nerve muscle preparation, but it was promptly realized how valuable it would be could one determine the excitability of the nerves and muscles in the intact human subject, and numerous attempts were made toward this end.

Weiss had already made determinations on the human subject, but his pistol method is too long and tedious ever to come into general use. Adrian¹² has also used the pendulum of Keith Lucas for this purpose, and Ströhl his égersimètre. Lapique developed his chronaximètre to be used in the clinic. In all of these the keys are a source of continual annoyance, and for this and other reasons they have been abandoned.

In this country Sachs and Malone² have described a spring driven instrument resembling the chronaximètre of Lapique, but in which the troublesome keys have been eliminated. This they have called a chronomyometer. The keys unfortunately have been eliminated at the expense of flexibility, for one is limited, by the construction of the apparatus, to five different times of passage of the current, unless one goes to the trouble of changing the contact plate. Sachs and Malone doubtless never intended it to show more than an approximation of the actual chronaxie, and it serves very well in the gross changes of nerves after section. In many neurologic conditions, however, more accurate

10. Lapique: Soc. de biol. **78**:695, 1915.

11. Ströhl: Jour. de radiol. et d'électr. **6**:375, 1922.

12. Adrian: Brain **39**:1, 1916.

determinations are necessary. For these it is also necessary to take into account the variations in the resistance of the patient.

Bourguignon¹³ has shown that all measures of the resistance of the subject by the aid of the galvanic current were illusory and could not serve for the short waves used in the determination of chronaxie. He was therefore driven to abandon the attempt to estimate the resistance of the subject, without which all measures of chronaxie were erroneous, and succeeded in eliminating it by a method originally devised by Lapicque, as we will show further on.

The sole method of determining chronaxie in the human subject, which, because of its rapidity and accuracy, is actually entering into the daily practice of electrodiagnosis, is the direct outcome of the work of Bourguignon, chief of the laboratory of electrotherapy at the Hospice de la Salpêtrière. Bourguignon¹⁴ makes use of condenser discharges and his method may be described briefly as follows:

The resistance of the patient is eliminated by putting in parallel with him a resistance of 10,000 ohms, the patient and 11,000 ohms in series with him being in the other branch. Four thousand ohms are in series with the system in the general circuit, before the bifurcation (Fig. 3). The circuit thus mounted has a resistance practically constant, no matter what region of the body is examined, its average value being 10,500 ohms. For this resistance it is necessary to use a source of at least 200 volts and nonpolarizable electrodes. It is then only necessary to find the capacity corresponding to the chronaxie and calculate the chronaxie by the formula already given.

With the circuit which Bourguignon uses, one can simplify the calculation, since the resistance is constant. If we express the capacity in microfarads instead of in farads, one microfarad will correspond to a chronaxie of 0.004, for $\tau = RC\tau \times 0.37$, then $\tau = 1^{mf} \times 10,500_{ic} \times 0.37 = 0.004$. One can therefore simplify the formula of Lapicque and write:

$$\tau = C\tau \times 0.004.$$

The chronaxie as determined by this method is identical with the figure determined by the pistol of Weiss (when the variation in resistance of the body is similarly eliminated), as I have had occasion to see verified time and again in Bourguignon's laboratory.³ It evidently gives results coinciding with those obtained by the Lucas pendulum. Adrian¹² gives for the tibialis anticus of the normal human subject, an average chronaxie of 0.00016. Bourguignon's figure for the same muscle is 0.00025, sensibly the same for such minute quantities. More-

13. Bourguignon: Acad. d. Sc., Feb. 14, 1916.

14. Bourguignon: Soc. de biol., April 30, 1921; La Médecine 3: February, 1922; Questions neurologique d'actualité, Masson et Cie, 1922.

over, there is reason to believe Adrian's figure inaccurate, both because of the manner of placing his electrodes, and also because the figure he gives for the nerve of the tibialis anticus is 0.00025. It is known that the chronaxie of a muscle and that of its motor nerve are identical. The method of Bourguignon is, therefore, accurate, and has the great advantage of being very rapid and easy of operation. It enables one to make rapid, accurate determinations and is capable of handling any range of variation of the chronaxie.

The method has one disadvantage; it requires the use of a source of 200 volts. This disadvantage seems to have been overcome in the posthumous work of Guilleminot,¹⁵ who has adapted the method for use on the ordinary 110 volt circuit. He makes use of two condensers, one with a capacity of 0.2 microfarad and the other with a capacity of 20 microfarads. A commutator allows one or the other to be used at will. The resistances are mounted as those of Bourguignon but are only of half the value. The instrumentation is so mounted that also by the use of a special key and manipulator one can make in succession an examination of the faradic excitability, an examination of the galvanic threshold, and finally a determination of the chronaxie with the subject in circuit with the resistances.

The method of operation is based on the formula developed in the foregoing:

$$\frac{a}{b} = RC \left[\frac{V_o}{V_{rh}} - \log_e \frac{V_o}{V_{rh}} - 1 \right]$$

Using a single condenser, with the resistance fixed, the product RC remains constant and V_o and V_{rh} can be taken as independent variables. One searches the rheobasic voltage by closing the hand key in the continuous circuit, then with the same current charging the single condenser, one searches the threshold of contraction at discharge on augmenting the voltage. The chronaxie may be calculated by the foregoing formula.

To avoid the complicated calculation, Guilleminot makes use of a slide rule, which is so constructed that, since the ratio $\frac{a}{b}$ depends not on the absolute values of V_o and V_{rh} but on the ratio $\frac{V_o}{V_{rh}}$, it reads directly the value of $\frac{a}{b}$ once V_o and V_{rh} are known, provided the product RC is equal to 1. With the system mounted as described the product RC is not 1 but 0.001 with the condenser of small capacity and 0.1 with that of larger capacity. One has, therefore, only to multiply the number read on the slide rule by 1000 or by 10 as the case may be. It is difficult to imagine an easier, more rapid or more effective method. It has been shown by Bourguignon to be sufficiently

15. Guilleminot: J. de radiol. et d'electr. 6:157, 1922.

accurate for clinical use, and for all ordinary clinical purposes its range is sufficient without surpassing the ordinary voltage of 110 volts.

VALUE OF CHRONAXIC DETERMINATIONS

It will be possible to give only a bare and incomplete outline of the astonishing results of the application of this method in the normal and pathologic human physiology.

Keith Lucas¹⁶ has shown that in the frog's muscle there are three substances with different excitation times which he calls the α , β , and γ substances. The β substance, which in the frog has a characteristic of excitability in the neighborhood of 0.0001, is doubtless in the human skeletal muscle too rapid to influence ordinary determinations. Adrian¹⁷ was unable to find any evidence of the β substance in the human muscle. The γ substance has been shown to be the nerve fibers and the α substance the muscle fibers.

When its motor nerve is intact, the normal human muscle gives the same chronaxie when excited by the nerve, the motor point or longitudinally. Probably in each case it is the γ substance which is excited. In a pathologic case, however, at the motor point the phenomena are very complex, owing to the fact that the pathologic muscle is heterogeneous and has at least two chronaxies, one smaller by excitation of the nerve and one larger, which is found by longitudinal excitation. In pathologic cases, therefore, the chronaxie should never be determined by stimulation at the motor point.

In pathologic conditions of the peripheral nerves in man, when the muscle is excited by the nerve, the chronaxie rarely surpasses five or six times its normal value; either there are still muscular fibers excitable by the nerve and the chronaxie does not surpass five or six times its normal value, or there are no muscular fibers excitable by the nerve, and the excitation of the nerve is not effective.

There is an intimate relationship between the rapidity and form of contraction and the size of the chronaxie, and in slight pathologic lesions the chronaxie reaches ten to fifteen times its normal value before any slowing of the contraction is visible. The length of the chronaxie varies from 0.00008 to 0.0007 for a normal muscle, to 0.01 to 0.07 in the case of a muscle of myotonic contraction.

It is quite evident from the work of Bourguignon, of Adrian and of Sachs and Malone that determinations of the chronaxie enable one to follow accurately the evolution of a pathologic process affecting the neuromuscular mechanism.

16. Lucas, Keith: *J. Physiol.* **36**:113, 1907.

17. Adrian: *Arch. Radiol. & Electroth.* **21**:379, 1916.

Bourguignon¹⁴ has attained astonishing results in his studies of the pathologic physiology. He has shown that the reflex atrophies following joint lesions are accompanied by alteration of the chronaxie of the muscles affected. Every hypertonic or contracted muscle has a chronaxie smaller than normal. Every hypotonic muscle has a chronaxie larger than normal. The attitude of the different segments of the limbs depends on the relation of the chronaxies of the anterior and posterior muscles. In the Parkinsonian syndromes following epidemic encephalitis, for example, with rigidity and bradykinesia, the normal difference between the anterior and posterior muscles of the limbs tends to disappear.

In any segment, the chronaxie is the same for all the muscles which act synergically. Nerves and muscles having the same chronaxies in a limb are attacked or respected by the same morbid agents, for example, in lead poisoning. The flexors have a chronaxie twice as small as the extensors. The synergic muscles of the proximal segment have a chronaxie from two to five times smaller than those of the distal segment.

For the study of the normal and pathologic physiology of the neuromuscular mechanism, the chronaxie determination has proved its value. Its practical value is not so evident. In traumatic nerve lesions its limitations have been clearly detailed by Adrian.¹⁷ The presence or absence of excitable nerve fibers peripheral to a trauma to a nerve trunk can be detected well enough by the ordinary faradic coil. In the determination of whether the cut end of a nerve is growing down toward the muscle or is blocked by scar tissue, Tinel¹⁸ has shown that there are several important signs of regeneration which appear before there is any change in the electrical reactions. As a rule, incomplete lesions are easily detected by sensory and other neurologic tests. In a few cases the nerve is much less excitable below the lesion than above it, and in this instance the response to short excitations is more readily obtained by stimulating above the site of the injury.

In the case of section or other injury to a nerve, determination of the chronaxie is, however, of value in following the condition of the muscle itself and determining the effect of our efforts to keep the muscle in good condition. In the diagnosis of primary affections of the muscles, such as myopathies and Thomsen's disease, the chronaxie may be found ten to fifteen times its normal value before any slowing of the contraction of the muscle is visible.

Because of the fundamental importance of the work of Bourguignon and his extensive experience, it seems fitting to close with an appreciation of the value of the method in his own words:

18. Tinel: *Les blessures des nerfs*, Masson et Cie., Paris.

From a practical point of view the chronaxie gives us a precise measure of the functional value of the nerves and muscles. It permits us to follow very closely the evolution of pathologic processes. It permits us to judge of the efficiency of our therapeutic measures. If it adds little to diagnosis, it is indispensable for the prognosis, to judge the evolution, and to determine with precision the therapeutic indications.

ARSPHENAMIN THERAPY IN STATE HOSPITALS FOR MENTAL DISORDERS *

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The treatment of syphilis in its varied manifestations as encountered in hospitals for the insane is a subject which has interested many physicians, not only those in our own specialty, but also many primarily devoted to other branches of medicine. This, of course, is indicative of its importance, and no one can gainsay the fact that it is a problem of the first magnitude. One finds the most widely diverging views as to the proper method of therapy, as well as to the kind of cases that should be treated, the scale running all the way from the enthusiast who is curing general paralysis, to those who see little or no good in any treatment. This is especially true in instances in which a patient has shown mental symptoms necessitating commitment to a state hospital.

We shall review briefly the literature, outline the plan which has been followed at Central Islip for the past year or so, present our results and our failures, and suggest for consideration a few conclusions.

PART I. REVIEW OF THE LITERATURE

The review of literature which we have made is much too long to be given in its entirety in this discussion. We have summarized this and present it for consideration under nine headings.

1. ARSENIC PENETRATION OF THE NERVOUS SYSTEM

Corbus and others¹ mention experimental work showing that the intravenous injection of hypertonic saline solution (15 per cent.) causes a rapid withdrawal of fluid from the brain. The restoration of fluid begins in about six hours; so they injected arspenamin intravenously six hours after the salt solution. They believe that the amount of arsenic recoverable from the spinal fluid in their cases was as great as

*Read at the Interhospital Conferences of the New York Hospitals, Hudson River State Hospital, Poughkeepsie, March 16, 1922, and Utica State Hospital, March 23, 1922, before the Brooklyn Neurological Society, May 17, 1922.

1. Corbus, B. C.; O'Connor, V. J.; Lincoln, Mary C., and Gardner, Stella M.: Spinal Drainage Without Lumbar Puncture; A New Method for Increasing the Penetration of Arsenic Into the Spinal Fluid in the Treatment of Neural Syphilis, *J. A. M. A.* **78**:264 (Jan. 28) 1922.

with any method suggested to the present time. Mehrtens and MacArthur² recovered arsenic in 43 per cent. of their patients treated intravenously; spinal drainage did not increase the number of penetrations, but if meningeal irritation was produced six hours before the intravenous treatment by the injection of a small amount of the patient's serum, the percentage rose to 92, and more arsenic was also recovered. This seems to us to have a bearing on results claimed for the Swift-Ellis treatment. We suggest that this method be tried out by anyone who has faith in the curability of late neurosyphilis with the present arsenic preparations. Morse³ believes that there is no reliable method for the quantitative estimation of arsenic in such small quantities. He prefers to discuss the matter in terms of concentration, and believes that if the rate of excretion from the central nervous system is equal to that from other tissues, one would expect at the end of a week to have a concentration of at least 1:800,000, still probably well within the lethal requirements. Therefore, in giving a course of treatment, the intervals between arsphenamin injections should not be longer than one week.

2. METHODS

From 1917 to 1919, Fordyce⁴ and Sachs⁵ carried on a wordy war in the medical journals with the intraspinal method pitted against the intravenous. Sachs condemned the intraspinal method, and in a personal communication received in January, 1922, he writes, "I am more opposed to intraspinal therapy than ever I was. I am perfectly frank to state that the intraspinal method has been exploited not always for purely scientific ends, and the dangers of it have never been honestly brought to the notice of the medical public." Kaliski and Strauss,⁶ who are associated with Sachs, agree with him in their papers published in 1918 and 1922. Fordyce,⁷ on the other hand, continues to be an ardent

2. Mehrtens, H. G., and MacArthur, C. G.: Therapy of Neurosyphilis Judged by Arsenic Penetration of the Meninges, *Arch. Neurol. & Psychiat.* **2**: 369 (Oct.) 1919.

3. Morse, Stern: Personal communication.

4. Fordyce, J. A.: Truth About Intraspinal Injections in Treatment of Spinal Syphilis—A Reply, *J. A. M. A.* **69**:1482 (Nov. 3) 1917.

5. Sachs, B.: The Truth About Intraspinal Injections in Syphilis of the Nervous System, *J. A. M. A.* **69**:681 (Sept. 1) 1917. On the Intravenous. Not the Intraspinal, Use of Arsphenamin in Syphilis of the Nervous System, *Arch. Neurol. & Psychiat.* **1**:277 (March) 1919.

6. Kaliski, D. J., and Strauss, I.: On Syphilis of the Nervous System, *Am. J. Syphilis* **2**:609 (Oct.) 1918. The Significance of Biologic Reactions in Syphilis of the Nervous System, with Notes on Treatment, Especially Intraspinal, *Arch. Neurol. & Psychiat.* **7**:78 (Jan.) 1922.

7. Fordyce, J. A.: The Importance of Recognizing and Treating Neurosyphilis in the Early Period of the Infection, *Am. J. Med. Sc.* **161**:313 (March) 1921.

advocate of the Swift-Ellis method. Solomon⁸ agrees with Fordyce and advocates also intraventricular injections when results are not obtained in other ways. Dercum,⁹ on the other hand, supports Sachs, and adds that the beneficial effects hitherto ascribed to the Swift-Ellis and kindred methods, are entirely due to the incidental spinal drainage. Dercum's ideas are supported by Schaller and Mehrrens.¹⁰ Stokes and Osborne,¹¹ who tried out the intraspinal method in a faithful manner, say that they are not convinced that it is of any value. Chargin¹² tried out different dosages and combinations with mercury in active systemic cases; he advocates what he calls a chronic intermittent method, that is, with the arsphenamin injections five days to a week apart, and a course to consist of six to eight injections of arsphenamin, and ten to twelve injections of mercury. His ultimate results were as good as with the more intensive methods, and the toxic dangers were avoided. It seems to be the consensus of opinion that in neurosyphilis moderate doses with reasonable intervals are safest. We think the experience of others justifies us in the plan we have followed at Central Islip.

3. ARSPHENAMIN OR NEOARSPHENAMIN

Rosanoff¹³ summarized the opinions of a large number of people with whom he corresponded, and stated that the majority favored neo-arsphenamin. Our review also shows that this is the general trend; and the majority of the New York State Hospitals now use the newer product. This preference seems to have a sound physiologic basis, Schamberg¹⁴ stating that while neo-arsphenamin is less active, the discrepancy can be made up by the size of the tolerated dose, and that it is less toxic and causes less biochemical disturbance of the blood and tissues. The time required for administration is, of course, an important point, especially in state hospitals. We recommend that when an arsenical is considered indicated in state hospital work, neo-arsphenamin

8. Solomon, H. C.: Clinic on the Diagnosis and Treatment of Neurosyphilis, Boston M. & S. J. **183**:723 (Dec. 23) 1920; *ibid.* **183**:768 (Dec. 30) 1920.

9. Dercum, F. X.: The Functions of the Cerebrospinal Fluid, with a Special Consideration of Spinal Drainage and of Intraspinal Injections of Arsphenamized Serum, Arch. Neurol. & Psychiat. **3**:230 (March) 1920.

10. Schaller, W. F., and Mehrrens, H. G.: Therapy in Neurosyphilis with Particular Reference to Intraspinal Therapy, Arch. Neurol. & Psychiat. **7**:89 (Jan.) 1922.

11. Stokes, J. H., and Osborne, E. D.: Relative Effectiveness of Various Forms of Treatment in Neurosyphilis, J. A. M. A. **76**:708 (March 12) 1921.

12. Chargin, L.: Antisyphilitic Therapy, J. A. M. A. **76**:1154 (April 23) 1921.

13. Rosanoff, A. J.: Some Practical Points in the Organization of Treatment of Syphilis in a State Hospital, State Hosp. Quart. **6**:319 (May) 1921.

14. Schamberg, J. F.: Arsphenamin Versus Neo-Arsphenamin, J. A. M. A. **73**:1883 (Dec. 20) 1919.

be used. We feel, however, that there is need of a drug more toxic to the spirochete and less so to the patient, also one which will penetrate better into the central nervous system.¹⁵

4. PERIOD OF INFECTION OF CENTRAL NERVOUS SYSTEM AND ITS THERAPEUTIC SIGNIFICANCE

Fordyce⁷ firmly believes that the involvement of the nervous system occurs during the period of general dissemination or not at all. In his first paper he stated that in the first year following infection the spinal fluid showed evidences of disease in 25 to 30 per cent. of cases, and in a recent paper he and Rosen¹⁶ stated that their figure has risen to 38 per cent. However, they admit that their material is somewhat selected. The 1922 paper of Kaliski and Strauss⁶ summarizes the literature bearing on this question, and we think tends to support Fordyce's conclusions, although Kaliski and Strauss themselves believe that a mere increase in cells and globulin with a transient Wassermann reaction, points only to a pia-arachnoid irritation, and that this is, as a rule, temporary and of doubtful significance. Herrick and Dannenberg¹⁷ worked with infectious diseases not syphilitic, and found cells and globulin increased in patients with so-called meningismus. Ravaut¹⁸ believes the early changes are transitory and in the nature of a septicemia. Additional evidence in favor of the belief in the early invasion has

15. Since this paper was written we have received a circular letter from the New York State Department of Health, recommending arsphenamin in preference to neo-arsphenamin. This letter quotes from a circular letter issued by the Surgeon-General of the United States Public Health Service under the date of April 26, said letter being an order to the medical officers of the Public Health Service and stating, "Your attention is called to the fact that neo-arsphenamine is a much less constant and less reliable preparation than arsphenamine. Not only do certain batches of all brands of neo-arsphenamine show a tendency to deteriorate with age, frequently with definite increase of toxicity, but there is a pronounced irregularity in the therapeutic activity of different batches of neo-arsphenamine, regardless of the source of the preparation. This variation may reach several hundred per cent., and as a result the physician may get a much less satisfactory therapeutic result than when arsphenamine is used, as the latter, regardless of brand or lot number, shows a more uniform therapeutic activity.

"These facts warrant the Bureau in urging that arsphenamine be given the preference over neo-arsphenamine wherever it is possible to administer the former. This choice should be made regardless of mere convenience of administration, in which respect neo-arsphenamine has the advantage."

16. Fordyce, J. A., and Rosen, I.: Laboratory Findings in Early and Late Syphilis, *J. A. M. A.* **77**:1696 (Nov. 26) 1921.

17. Herrick, W. W., and Dannenberg, A. M.: Observations on the Cerebrospinal Fluid in Acute Disease, *J. A. M. A.* **73**:1321 (Nov. 1) 1919.

18. Ravaut: *Presse méd.* **27**:573, 1919.

been quoted by Solomon.¹⁹ The most important point made by Fordyce and others believing as he does is that the time to treat neurosyphilis is in the first year; and it is Fordyce's belief that it will require a long time before these early infections are systematically and properly treated. He lays stress on the improper facilities in public clinics and the lack of routine lumbar punctures.

5. DIAGNOSIS

In discussions on the treatment of neurosyphilis, so-called borderline cases are frequently stressed, and references are made to Dunlap's²⁰ paper on this subject. There is no question but that the various parenchymatous and mesoblastic changes at times are so equally divided that anatomic classification is difficult or impossible. Nevertheless, Dunlap in his paper states that the groups are ordinarily distinct, and we feel that the cases admitted to state hospitals are, as a rule, clear-cut clinically. The next thing to be considered in making a diagnosis is the serology. Southard and Solomon²¹ state that theoretically all tests may be negative and neurosyphilis still be present. It is apparently generally recognized that the spinal fluid may be negative in those cases in which the disease affects almost solely the blood vessels. That entirely negative fluids, that is, negative Wassermann and globulin tests, a normal cell count, etc., occur in other types, seems to us questionable, or at least rare. We did not find reports of any such cases with proper anatomic backing. Published reports of benefits obtained from therapy are frequently invalidated by the number of cases included with primarily negative blood and fluid tests.

6. EVALUATION OF SEROLOGIC CHANGES UNDER TREATMENT

Many workers lay great stress on the serologic changes occurring during arsphenamin therapy. We have seen so many striking fluctuations in the same persons with or without treatment, that we believe extreme caution should be used in weighing them. It is, of course, generally admitted, as stated by Morse,²² that the Wassermann test is a quantitative relationship between numerous variable factors, both in the specimens and outside of it. Morse thinks that the errors are mostly toward negative results. Of interest in this connection is the case of A. W., who was first admitted to Central Islip in 1916, at the age of 38. Since then he has twice been a patient at this hospital; all attacks have

19. Solomon, H. C.: A Review of Recent Literature on Neurosyphilis, *Arch. Neurol. & Psychiat.* **7**:235 (Feb.) 1922.

20. Dunlap, C. B.: Anatomical Borderline Between the So-Called Syphilitic and Metasyphilitic Disorders in the Brain and Spinal Cord, *Am. J. Insanity*, Special Number **69**:1045, 1913.

21. Southard, E. E., and Solomon, H. C.: *Neurosyphilis*, Boston, W. M. Leonard, 1917.

22. Morse, S.: *New York State Psychiat. Bull.* **2**:239 (April) 1917.

been of manic-depressive symptomatology, the last following a circular course. He has been at home as long as two years, working efficiently. In 1916, his blood test was reported + + + +. In 1917, he is said to have received one intraventricular treatment, one or more intravenous injections of arsphanamin and some mercury. Since then he has received no treatment. His spinal fluid in 1917 is said to have shown 72 cells and a + + + + Wassermann reaction. Recently two punctures have shown 6 cells and the spinal fluid Wassermann test persistently negative. Blood tests, however, have ranged from + + + + to negative; and blood drawn the same day has been reported positive from one laboratory and negative from another. Since 1916, he has shown some pupillary abnormality and unequal knee jerks.

7. LITERATURE FAVORING TREATMENT OF LATE NEUROSYPHILIS,
INCLUDING GENERAL PARALYSIS

We reviewed in detail ten or more papers advocating the treatment of paretic patients, but we believe that the authors did not prove their case. Cotton,²³ six years ago, claimed benefit in 58 per cent. of his cases, more than half of which he said had been definitely arrested. One of us recently visited the Trenton State Hospital and found that Dr. Cotton is now only slightly interested in the treatment of neurosyphilis. Solomon,⁸ in his earlier writings, makes such statements as, "Every paretic, except those in an advanced stage, should be treated." However, in his latest publication,²⁴ his statements are much more guarded. Campbell²⁵ refers to the fact that in the past repeated injections of tuberculin or nucleic acid gave rise to claims of success comparable with those now made for arsphenamin therapy. He says, "Although there is considerable difference of opinion it seems established that intensive treatment of general paralysis is of undoubted benefit in a certain number of cases." However, later in his article he adds, "It is important to remember that one is dealing with a serious progressive deterioration of the brain, the permanent arrest of which is very problematical." Amsden,²⁶ in 1914, was the first to bring the subject in its entirety before the New York State Hospital group. At first, he thought that he was securing good results. In 1918,²⁷ he reported on thirty-five cases with 48.5 per cent. of remissions, but in a personal communication received in December, 1921, he stated, "During the last five years we have not

23. Cotton, H. A.: The Treatment of Paresis and Tabes by Salvarsanized Serum, *Am. J. Insan.* **72**:485, 1916.

24. Solomon, H. C.: A Review of Recent Literature on Neurosyphilis, *Arch. Neurol. & Psychiat.* **7**:235 (Feb.) 1922.

25. Campbell, C. M., in *Nelson's Loose Leaf Medicine*, Thomas Nelson & Sons, 1920.

26. Amsden, G. S.: *New York State Hosp. Bull.* **7**:538, 1914.

27. Amsden, G. S.: *New York State Hosp. Quart.* **3**:334, 1918.

been doing any intraspinal treatment of paresis in any aggressive way." He thinks that the remissions obtained are somewhat more frequent, somewhat more prolonged and possibly of better character.

8. ADVISABILITY OF TREATMENT IN LATE NEUROSYPHILIS DOUBTFUL

Mitchell¹⁰ of Warren, Pa., in a discussion stated that they had been treating about fifty parietic patients annually for the last ten years, and while they thought they secured a larger percentage of remissions, the majority had been returned to the hospital. As a result of his experience, he regarded any improvement as temporary. Jackson and Pike²⁸ state that in fifty patients whose condition was diagnosed as parenchymatous neurosyphilis, to whom intensive treatment was given "intravenously, intraspinaly and intraventricularly supplemented by mercury and iodides, no lasting results were obtained, and each patient finally succumbed to the disease." Sheehan²⁹ believes that we are not yet in a position to state the ideal treatment for syphilis. He does not favor the treatment of latent systemic cases, neither do Jackson and Pike. Sheehan considers the prognosis hopeless in paresis and tabes, and that treatment may be contraindicated from a social standpoint.

In our review we found many more in favor of treating late neurosyphilis, including parenchymatous types, than opposed. However, as previously stated, we do not think that therapy as now practiced rests on a sound basis; there seems to us to have been a rush into expensive and intensive wholesale treatment which is not justified by preceding experience with the class of cases admitted to state hospitals.

9. LATENT SYPHILIS

In the literature one constantly encounters the terms "systemic syphilis," "latent syphilis," "neurosyphilis," etc., and we have divided our patients into two groups: those with systemic syphilis and those with neurosyphilis. In the systemic group we placed those with secondary lesions, those with a history of recent infection and those who showed no signs of syphilis except a positive Wassermann test of the blood. We, of course, realize that this division of our material is purely arbitrary. Latency, Wassermann-fastness, etc., are terms with clinical value, but the pathology is not known. Engman³⁰ states that a continued persistent positive Wassermann reaction usually signifies strains with neurotrophic or vascular affinities. However, we do not

28. Jackson, J. A., and Pike, H. V.: Interpretation of Wassermann Reaction of Blood Serum in Mental Diseases: Indications for Antisyphilitic Therapy, *J. A. M. A.* **76**:360 (Feb. 5) 1921.

29. Sheehan, R.: Remarks on the Neurology of Syphilis, *Mil. Surgeon* **49**: 252 (Sept. 21) 1921.

30. Engman, M. F.: *Nelson's Loose Leaf Medicine*, Thomas Nelson & Sons, 1920.

know where the focus is in our unimproved patients. We cannot say that it is not in the central nervous system; we will hazard no guess as to what later pathologic studies may show in these cases; neither can we assume with Engman that our unimproved systemic patients harbor strains considered resistant. Another alluring speculative possibility is organ inferiority inasmuch as all our patients are insane. Another possibility is infection with a weak or nonvirulent strain, but this entire subject is unknown. Engman and other writers assume that in latent syphilis either changes take place in the pathogenic activity of the organism, or there is an increase in the defensive forces of the cells, and that these changes presumably occur because of the elaboration by the body of immune substances. Just when the production of these stops, its relation to the death of the last spirochete, and the relation of these bodies to the Wassermann reaction, are all speculative questions. It has been stated that a positive Wassermann test indicates that the person is harboring live organisms. They are not always present because they have been demonstrated in a few cases, neither can it be said that the individual does not go on producing antibodies or whatever they are for months or years after all the spirochetes are dead. Ebersson,³¹ commenting on the finding of spirochetes in semen and lymph glands in cases considered latent, says that because the organisms are found they need not be looked on as evidence of existing disease. Warthin³² has demonstrated postmortem spirochetes in practically every body organ and tissue of persons who during life gave a negative Wassermann reaction. He says that these organisms morphologically intact may rest there inactive indefinitely; and the fact that in other cases they cause something which gives a positive Wassermann reaction does not prove that they will ever be active in any other way. Conversely, the Wassermann-producing substance may not be present in sufficient quantity to give a positive reaction even when the disease is active clinically. All writers in discussing latent syphilis speculate as to the advisability of treatment. Some feel that there is danger of lighting up an old quiescent process, and nearly all of them refer to the person, frequently encountered, whose infection occurred thirty or forty years before, whose blood is + + + +, but who has throughout the entire period enjoyed robust health. These cases always raise the problem of strains, virulence of the organism and natural resistive powers of the host; and Engman says that there is no doubt but that certain strains always portend a grave prognosis. Conversely, there may be strains which injure the person little or not at all, and we feel

31. Survey of Literature: Syphilis, Nelson's Loose Leaf Medicine.

32. Warthin, A. S.: The Persistence of Active Lesions and Spirochetes in the Tissues of Clinically Inactive or "Cured" Syphilis, *Am. J. M. Sc.*, 1916, p. 508.

that the treatment of in old asymptomatic cases should always be approached with caution and reservations. Some syphilologists that we have talked with say that they usually discontinue treatment if no results are obtained after three courses. Engman says it is his experience that if a negative Wassermann reaction does not occur after the second course, it is extremely difficult to obtain. Our own cases bear this out, and also shown that serologic improvement tends to occur after the first course of treatment if it occurs at all.

PART II. THERAPY IN NEW YORK STATE HOSPITALS

In 1918, the New York state hospitals for mental disorders were circularized by Cheney, and at that time little treatment was being given. Since then there seems to have been a steady but slow increase in the number of patients treated. We circularized the twelve other civil hospitals inquiring about the number of patients treated during the fiscal year ending June 30, 1920, compared with those treated during 1921: in general, it can be said that there has been a considerable increase in the numbers treated during the last year. Combining the figures for the past two years, we have 543 patients treated more or less intensively intravenously, most of them selected cases of neurosyphilis with a few of systemic involvement. Five hospitals used old arsphenamin during the period under discussion, but two of these five are now using neoarsphenamin. Only one reported using iodids, but all except one used mercury. One hospital is now using spinal drainage. Nearly all report their general impressions in neurosyphilitic cases as serologic improvement common, general physical improvement fairly constant at least for a time, mental improvement questionable. One hospital reports a tendency to change the diagnosis to cerebral syphilis when improvement occurs in a case thought to be general paralysis; this has been mentioned in several articles. In connection with these reports from the various state hospitals, it is of interest to analyze certain figures given us by Dr. Pollock, statistician to the New York State Hospital Commission (Table 1).

These figures show little change in the number of paretic patients admitted during the last twelve years; the maximum number, 913, were admitted in 1918, and the minimum, 719, in 1912, with 827 during the last fiscal year—a gain of seven over 1920. The percentage discharged as improved and much improved is more interesting. The highest percentage, 17.8, was discharged in 1912; the lowest, 8 per cent., in 1917. In 1920, we see a decided improvement in the discharge rate, but it is still short of that of 1912. This, of course, might be interpreted as an expression of the more energetic and generalized treatment given in 1920; but with still more treatment in 1921, and the fact that we would naturally assume that results from treatment in 1920 would

be registered in 1921, we find instead of an increase, a fall to 12.4 per cent. Although some of the hospitals report the general impression that there are more remissions, this impression is not borne out by the figures of Dr. Pollock; and although some report that they tend to change the diagnosis from general paralysis to cerebral syphilis when marked improvement occurs, a study of the figures for cerebral syphilis does not bear this out. The high point of fifty-four in 1920 was equaled

TABLE 1.—GENERAL PARALYSIS IN NEW YORK STATE HOSPITALS

First Admissions		Discharged as Improved and Much Improved	
Year	Totals, Male and Female	Number	Percentage to First Admission
1910	815	108	13.3
1911	758	98	12.9
1912	719	128	17.8
1913	768	105	13.7
1914	774	96	12.4
1915	814	108	13.3
1916*	640	59	9.2
1917	866	69	8.0
1918	913	90	9.9
1919	880	85	9.7
1920	820	127	15.5
1921	827	103	12.4

* Due to change in fiscal year, only 9 month period reported.

CEREBRAL SYPHILIS CASES IN NEW YORK STATE HOSPITALS

First Admissions		Discharged Recovered		Improved and Much Improved		Total Percentage Benefited
Year	Number	Num	Percentage	Number	Percentage	
1919	26	5	19.2	9	34.6	54
1920	54	3	5.5	9	16.6	22
1921	38	4	10.5	13	34.2	45

TOTAL NUMBER OF PATIENTS WITH GENERAL PARALYSIS AND CEREBRAL SYPHILIS DISCHARGED AS BENEFITED (THIRTEEN CIVIL HOSPITALS)

Year	Total First Admission	Total Number Discharged Benefited	Percentage to First Admission
1919	906	99	10.9
1920	874	139	15.9
1921	865	120	13.8

in 1913, and in 1921 it dropped back to thirty-eight. Combining the figures for general paralysis and cerebral syphilis, we find 10.9 per cent. discharged as benefited in 1919, 15.9 per cent. in 1920; but 13.8 per cent. in 1921, a loss of 2 per cent.

That the long remissions quoted by some authors as attributable to therapy must be received with scepticism, is indicated by another table prepared for us by Dr. Pollock but not included in this communication, which showed the length of time out of the hospital for patients dis-

charged between 1910 and 1915 and later readmitted. The length of time out included time on parole. Two hundred and fifteen have been readmitted. Of these, twenty-five were out less than six months, but sixty-seven were out from six to twelve months, seventy-two from one to two years, twenty-seven from two to three years, nine from three to four years, eight from four to five years, and seven more than five years. The average length of time was a little more than one and a half years. We think that this remission rate as expressed by discharges must be borne in mind when we note that those treating large numbers of patients in active clinics, such as Solomon, report only one or two or three showing very good results, and seem to refrain carefully from giving the total figures of those treated. We also call attention to the fact that these figures are only for patients discharged and later readmitted, and do not include the large number paroled and returned.

PART III. PROCEDURE AT CENTRAL ISLIP HOSPITAL

In January, 1920, the state board of health began supplying us with arsphenamin, which they continued to do for more than a year. Since then we have used the product of the Dermatological Research Laboratory of Philadelphia, which the state board of health said most nearly resembled their own.

Our purpose was to treat: 1. All patients with so-called systemic syphilis, that is, those having a ++ or more Wassermann reaction of the blood, or clinical symptoms and with negative findings in the spinal fluid and negative neurologic signs. When the Wassermann reaction was only ++, it was confirmed by a second test before beginning treatment. 2. Those with cases diagnosed as nonparenchymatous cerebral syphilis. 3. Patients with general paralysis were treated only for special reasons, such as (*a*) those thought to have the early or tabetic type with little brain involvement; (*b*) those who had previously received intensive treatment; (*c*) those whose relatives insisted on treatment. The cases selected, then, were those most favorable for treatment, and included only for some special reason cases with a bad prognosis. This is a significant feature of the cases reported and should be borne in mind in examining the results.

The methods to choose from in administering the arsenicals were many, and the opinions as to their value varied widely. It was decided to adopt a simple, well recognized method and to persevere with it until our results or the findings of others should warrant a modification or change. The administration of arsphenamin intravenously and of mercury intramuscularly was the procedure adopted.

The time consumed varied. It depended chiefly on the ease with which a needle was properly inserted into a vein. Matters were expedited by setting up a second infusion apparatus so that two phy-

sicians could work together. Eleven patients was the greatest number treated at one session, and it required an hour and a half or two hours to treat these when things were running smoothly. When difficulties with the veins were encountered, this time might be extended an hour or two. The amount of time consumed by this method, in which each patient receives 150 c.c. of solution, is a great disadvantage when there are a large number of patients to be treated.

No dangerous reactions followed the administration of arspenamin, except in one case. Milder reactions varied. Some patients showed none at any time, while others were upset only occasionally. In a few cases we believe errors of technic were responsible. While on the table, the only untoward symptoms noted were nausea and vomiting at the completion of a dose. On one occasion, two patients in succession manifested nausea, and it was believed that insufficient alkalization was responsible. After that we added an excess of alkali, and we had no further disturbances. On one occasion, most of the patients treated had a rise in temperature and complained of gastric distress, the temperature of one patient reaching 105 F. The next morning they all appeared none the worse for their experience. On this occasion it was thought that the water used was not freshly distilled. Several patients developed an inflamed condition of the skin of the arm with exfoliation. A dressing of aluminum acetate, followed in a few days by zinc oxid ointment, usually cleared up the skin. The conditions were evidently due to leaking of the arsenic solution into the tissues around the vein. One patient had considerable induration, and his arm was massaged for several weeks. Another patient who had shown some nausea after each dose of his fourth course, had this inflamed condition at the bends of both elbows. The administration of arspenamin being continued, he then had numerous patches of exfoliative dermatitis on his body. One patient developed an intense exfoliative dermatitis after his third dose. This began over his ears and spread over his body. His condition became critical. He ran a temperature ranging as high as 105 F. for more than a week. A general furunculosis was superimposed, and he still had large abscesses in the axilla a month later. The condition finally cleared up.

We had considerable difficulty in obtaining a suitable mercury preparation. We experimented with several, and finally adopted the ampules of mercuric salicylate as supplied by any of the wholesale drug firms. We found this suspension entirely satisfactory from both mechanical and physiologic standpoints, the latter as shown by salivation. We found that we could seldom give more than 1 grain weekly for the full course of ten weeks. At times the mercury was given coincident with the arsenic preparation. Again one course followed the other. Scham-

berg,³³ in recommending that the mercury follow the arsenic, says, "Vigorous mercurial treatment is often responsible for arsenical intoxication." Our two patients with exfoliative dermatitis were taking both arsenic and mercury at the time this condition developed.

TREATED SYSTEMIC CASES

The results of treatment may be discussed under four headings:

1. *Wassermann Results* (Table 2).—There were twenty-three cases in this group. The courses given ranged from one to four, each course consisting of five or six doses of arsphenamin and ten doses of mercury. Dividing these cases according to the results of the Wassermann test, we have four groups.

Group A, Negative: This group consists of all cases in which a persistent negative Wassermann reaction was obtained. Nine cases became negative, but in three of these a positive reaction again appeared, leaving six cases in this group. Five of these, Cases 1, 2, 4, 5 and 7 became negative after one course of treatment; one, Case 3, after two courses.

Group B, Reduced: This group consists of all cases in which there was a reduction in the degree of positiveness of the Wassermann test but without a negative reaction. There was a slight reduction in four. Three cases, Cases 6, 14 and 15, were reduced after one course; one, Case 11, after three courses; and in all four the change was only 1 degree.

Group C, Not Reduced: This group consists of some unchanged cases, and of others showing changes in the Wassermann reaction, but without permanent reduction. There were eleven cases in this group. Three cases, Cases 10, 13 and 20, were not reduced after one course; three, Cases 16, 17 and 19, were not reduced after two courses; two, Cases 8 and 9, were not reduced after three courses, and three, Cases 12, 18 and 21, were not reduced after four courses.

Cases 12 and 21 are interesting because the blood became negative after the first course, positive after the second course, and again more positive after the third and fourth courses. As Case 12 had only a + blood test before treatment, one might ask whether a quiescent latent syphilis was not roused to renewed activity by the treatment.

Group D, Died: Two of the patients died within six weeks of the last dose, without tests being made, one of tuberculosis and one of enteritis.

2. *Clinical Improvement Compared with Scrologic Results*.—In this group of systemic cases, clinical improvement could in general be

33. Schamberg, J. F.: Clinical Commentary on Studies of Histologic Changes in Organs Induced by Arsphenamins, by Neo-Arsphenamin and by Mercury. *Arch. Dermat. & Syph.* 3:571 (April) 1921.

TABLE 2.—SYSTEMIC CASES *

Case	Age	History	Previ- ously Treated	First Test		After 1 Course, Blood	After 2 Courses, Blood	After 3 Courses, Blood	After 4 Courses, Blood	Final Test, Blood	Months Observed	Outcome
				Blood	Spinal Fluid							
1	30	+	..	+++	..	—				—	10	P
2	25	+	+	+++	..	++				—	9	P
3	20	+	—	+++	..	++	—			—	16	P
4	42	+	—	+++	..	—				—	10	P
5	39	+	+	+++	..	—				—	18	P
6	43	+	+	+++	..	+				+	9	P
7	23	—	—	+++	..	—				—	10	P
8	27	+	+	+++	..	++	++	++++		++++	11	H
9	36	+	+	+++	..	++	++	++++		++++	17	H
10	21	—	—	+++	..	+++				+++	30	P
11	29	—	—	+++	..	+++	+++	+++		+++	50	H
12	41	—	—	+++	..	+++	+++	+++		+++	27	H
13	31	—	—	+++	..	+				+	18	H
14	50	—	—	+++	..	+++				+++	15	H
15	46	—	—	+++	..	+++				+++	15	H
16	48	—	—	+++	..	+++	+++			+++	19	H
17	34	—	—	+++	..	+++	+++			+++	19	H
18	27	+	—	+++	..	+++	+++	+++		+++	8	H
19	45	+	+	+++	..	+++	+++	+++		+++	24	H
20	38	+	+	+++	..	+++	+++	+++		+++	26	H
21	45	+	—	+++	..	+++	+++	+++		+++	10	H
22	45	—	—	+++	..	—	+	++		+++	27	H
23	58	—	—	+++	..						4	D
24		—	—	+++	..						3	D

* In this table and in Table 4, + in history column means "history of syphilis obtained"; — means "not obtained." + in previous treatment column means "history of previous treatment"; — means "none." All other + and — signs refer to Wassermann tests. In outcome column: P means parole; H, still in hospital; D, died.

correlated with a reduction in the positive findings in the blood Wassermann test. In other words, as shown in Table 3, patients with a reduction in the degree of positiveness of the Wassermann reaction were more often the ones showing mental or physical improvement or both, or vice versa.

Summarizing these, we find clinical improvement in seven cases out of ten that showed a reduction in the Wassermann test. Four patients improved clinically out of eleven that showed no reduction in the Wassermann test.

3. *Age of Infection Compared with Results of Wassermann Test.*—The effect of treatment in relation to the duration of the disease deserves consideration.

We find that in our cases a reduction in the Wassermann test was more frequent in those of short duration. It occurred in all three cases of less than one year's duration. In cases ranging from three to twenty

TABLE 3.—CASES OF SYSTEMIC SYPHILIS *

Group	Results of Blood Wassermann Test	Mental and Physical Improvement	Mental Improvement Only	Physical Improvement Only	Unimproved	Died	Total
1	Reduced to —	2-4-5-7		3-1			6
2	Reduced.....			11	6-14-15		4
3	Not reduced.....	10	12	13-20	8-9-16-17-18-19-21		11
4	Not tested.....					22-23	2
	Total.....	5	1	5	10	2	23

* Figures in all except totals refer to case numbers.

years' duration, the exact length of time the syphilis had existed seemed to be of little moment, Case 18, of six years' duration, showing no reduction after four courses, and Case 4, of twenty years' duration, maintaining reduction after one course.

4. *Effect of Previous Treatment.*—There was a history of previous treatment in seven cases. The amount of this treatment varied. Most of the patients treated had received mercury and some of them arsphenamin. In none was the treatment considered intensive. Reduction in the Wassermann test was obtained in cases of short duration, but no influence of previous treatment was observed in cases of long duration.

CASES OF TREATED NEUROSYPHILIS

1. *Serologic Findings Following Treatment* (Table 4).—There were fifteen cases in this group. Of nine cases in which both the blood and spinal fluid were retested, two, Cases 1 and 4, showed a decreased Wassermann test in both, with a reduced cell count. One case, Case 12,

TABLE 4.—CASES OF NEUROSYPHILIS

[illegible]

showed a reduction in cells and in the spinal fluid Wassermann test, but no change in the blood. Cases 2, 3, 8, 9, 10 and 13 showed a reduction only in cells. Of five cases retested only in the blood, three, Cases 5, 11 and 14, showed a reduction; in the other two there was no change. None reached and maintained a negative Wassermann test in either the blood or spinal fluid, except one case, Case 11, in which the patient died.

2. *Clinical Improvement Compared with Serologic Findings* (Table 5).—Mental and physical improvement were noted in three cases, Cases 1, 2 and 7. Case 1 showed reduction in the Wassermann test of both the blood and spinal fluid and a reduced cell count after only three doses of arsphenamin and five of mercury. The patient in Case 2 showed no reduction serologically after two courses in the hospital;

TABLE 5.—CASES OF NEUROSYPHILIS *

Group	Serologic Results	Mental and Physical Improvement	Mental Improvement Only	Physical Improvement Only	Unimproved	Died	Total
1	Reduction in blood, spinal fluid and cells.....	1			4		2
2	Reduction of spinal fluid and cells.....					12	1
3	Reduction of cells only.....	2		3	8-9-10	13	6
4	Reduction of blood only.....				5	11-14	3
5	Not reduced.....	7			6		2
6	Not tested.....					15	1
	Total.....	3	0	1	6	5	15

* Figures in all except totals refer to case numbers.

he improved clinically and was paroled. He is receiving further treatment in a New York City clinic, and on the last examination outside the hospital, the cells were reduced to 1, while the spinal fluid was + + + + and the blood continued —. In Case 7, the blood was not reduced after one course; the spinal fluid has not yet been reexamined. He is still receiving treatment in the city clinic. Physical improvement alone was noted in Case 3. The patient showed no reduction of positiveness in the blood or spinal fluid after one course of treatment here; he was paroled and received six intramuscular, six intravenous, eight intraspinal and one intraventricular treatment outside. The spinal fluid continued to be + + + +; the last cell count was 50 as compared with 65 before treatment.

3. *No Clinical Improvement and Death of Patient*.—Four patients, Cases 5, 6, 9 and 10, were unimproved after one course of treatment. One of these, Case 5, showed reduction in the Wassermann test of the

blood, and two, Cases 9 and 10, showed a reduction in cells. However, in Case 10 both counts were within the normal limits. One case, Case 6, showed no change.

There was no improvement in Case 4 after two courses of treatment by us. There was a reduction in the cells and in the blood and spinal fluid Wassermann tests. This case is of interest because of previous treatment. The patient had spent two and a half years in another hospital, where he received nine intraventricular injections as well as sixteen intravenous and twelve intraspinal injections. Just prior to his discharge from the other hospital, tests showed only 4 cells, but the globulin test was + and the gold chlorid 5554300000; the Wassermann test was not reported. Under treatment in Central Islip, the cells were reduced from 318 to 26, the spinal fluid from + + + + to — and the blood from + + + + to + +. In view of our findings in all cases, we attach no importance to these changes. The other case, Case 8, was complicated by the presence of a bullet in the temporal fossa. The patient's spinal fluid varied at different times from — to + + + +. The cells were fairly constant for tests made over twelve years, ranging from 40 to 60, and did not change after three courses of treatment until after the removal of the bullet, when they were reduced to 12, both the spinal fluid and blood Wassermann tests at this time being + + + +.

Five patients died; three, Cases 11, 14 and 15, after one course, and two, Cases 12 and 13, after two courses. The patient in Case 12 showed a reduction to + + in the spinal fluid test and a reduction in cells from 196 to 14. In Case 13 both blood and spinal fluid tests were + + + +, with a reduction of cells from 77 to 20. Both patients died within six weeks of the last dose. In Case 11 there was a — Wassermann test in the blood after one course. The patient in Case 15 died without retests, and in Case 14 the blood was reduced only 1 degree, the spinal fluid not being retested.

In this group, then, we find little correlation between the reduction serologically and clinical improvement, a striking contrast when compared with the systemic cases (Tables 3 and 5).

Variability of Findings in Both Groups of Cases.—In most of the cases more tests were made than are recorded in the tables, and frequently a negative or + would appear in cases in which the result was regularly + + + +. This cannot in any way be correlated with the treatment, as it occurred in some cases before treatment, and a positive result might follow a course immediately, to be succeeded by a negative result a few months later, or vice versa, without any uniformity or apparent reason. On this account many patients had more than one Wassermann test made between courses, and at

times findings of another laboratory were used in checking up reports. This held true of the spinal fluid as well as the blood, and cell counts were found to vary within a wide range as well.

PART IV. CONCLUSIONS

1. The division of our case material into systemic and neurosyphilitic is purely arbitrary, but conforms to the present accepted nomenclature. We realize that in our old asymptomatic cases we do not know where the focus of disease or of spirochetes is; in fact, we do not know that there is any disease, or even that there are live organisms.

2. Speculation as to strains, virulence, natural body resistance, Wassermann-fastness, latency, etc., would be premature and has not as yet a sound pathologic and clinical basis.

3. Granting that the foregoing limits the scope of our conclusions, we feel that the literature can be summed up and correlated with our own material, and that certain definite recommendations and impressions can be stated.

4. Arsenic penetration of the central nervous system is said to be much increased by inducing a sterile meningitis and may be the mechanism accounting for the benefits attributed to the Swift-Ellis treatment.

5. We think it is conceded that about one third of all cases of syphilis show changes in the spinal fluid in the early stage of the infection, and that about 10 per cent. develop frank serious involvement of the nervous system.

6. Until there is better treatment of syphilitic patients in the early stages, and until a radical change occurs in the methods followed by metropolitan clinics and physicians in general, we do not look for any material reduction in the number of late cases needing institutional care.

7. We question very much the reliability of reports giving results of treatment in neurosyphilis with primarily entirely negative spinal fluid, and we call particular attention to the general lack of necropsy material. We also question the therapeutic significance of varying degrees of positiveness of the Wassermann reaction, but are not in a position to draw any conclusions along these lines as to the gold chlorid test. We do believe that the gold chlorid test should be performed in all cases.

8. As to methods: We believe that the consensus of opinion is that in systemic cases definite courses of an arsenic preparation and of mercury should be given; that the arsenical should be given in moderate doses and not more than one week apart; that to effect a cure in even early systemic cases prolonged treatment should be given. As to neurosyphilis: We believe that the multiplicity of methods is merely indicative of the seriousness of the disease and impossibility in general of

affecting it favorably. We feel that none of the direct methods so far advanced has solved the problem, and our review of the literature has not convinced us that their advocates have proved their case. We are impressed with the experimental work in inducing meningeal irritation and would like to see more done along this line.

9. Our experience, although in only two cases, bears out Schamberg's statement that the toxicity of the arsenical is increased by giving mercury at the same time. We recommend that it be given as a separate course following the course of arspenamin.

10. In the so-called latent systemic cases as encountered in state hospitals, unless serologic improvement occurs after the first course, none is to be expected; and if this improvement is only a slight reduction in the degree of positiveness, it is of little significance.

11. In only two of our cases in the systemic group was syphilis thought to have a direct bearing on commitment; nevertheless, clinical improvement could, as a rule, be directly correlated with serologic improvement.

12. Our material shows that when the duration of the systemic syphilis is one year or less, therapeutic response is to be expected; after three years it is much less certain.

13. In our neurosyphilitic group, all patients who were retested showed a reduction in cells. None reached and maintained a negative Wassermann reaction in either the blood or the spinal fluid, except one that died.

14. Five improved sufficiently clinically to warrant parole; however, in only two patients, or 14 per cent., could treatment have been of any influence, which is little better than the remission rate for the entire state.

15. Our neurosyphilitic group does not show the correlation between clinical and serologic changes that the systemic does; two cases closely resembling each other in the period since infection and the nature of the process clinically progressed rapidly to death, although one showed well marked improvement in the blood and spinal fluid findings.

16. Although the literature contains numerous warnings in discussing therapy in the latent cases, we believe that our own material justifies at least one course, which must, however, be carefully given and the effects watched. Each case of this kind must be judged on its own merits with due regard to the age of the patient, general bodily health, past history, nature of the psychosis, etc., and continued treatment governed by results.

17. Although the majority of the authors reviewed favor treatment of neurosyphilis, we feel that nearly all of their reports are open to serious criticism; and many who advocate treatment of the paren-

chymatous types admit their lack of success. It is also to be noted that some of those formerly enthusiastic have greatly receded from their earlier position.

18. As our case material tends to show that prolonged conscientious intravenous treatment does not, as a rule, clear up old latent supposedly systemic syphilis, judging by the blood Wassermann reaction, on theoretical grounds alone, we would feel hopeless as to its efficacy in nervous system involvement. We feel that there is need of a drug more toxic to the spirochete and less so to its host.

19. Although our present material includes only cases in which intravenous therapy was employed, we nevertheless believe that in general, parietic patients should not be treated, at least not those admitted to state hospitals. We also believe that patients suffering from what is designated as late cerebral syphilis should usually not be treated, and that the time to treat neurosyphilis is during the first year or so after the primary infection.

20. We do not believe that the so-called borderline cases should be stressed from a therapeutic standpoint; that, on the contrary, the cases admitted to state hospitals are usually clear-cut clinically.

21. During the last two fiscal years there has been a decided increase in the number of syphilitic patients treated in the New York state hospitals; this has not been reflected in the discharge rate.

22. The economic side has at times been stressed, but we fail to see the economy when at best all that is produced is a remission; and to the expense of drugs must be added the expense of total longer care.

23. We place no reliance on one negative Wassermann reaction in a case under treatment; and, in view of the numerous discrepancies and contradictions which we have seen in the serology, we recommend repeated examinations in all unclear cases, and especially examination in more than one laboratory.

SCHIZOPHRENIC CATATONIA WITH ASSOCIATED METABOLIC AND VEGETATIVE FEATURES *

REPORT OF A CASE

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AND

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The case here reported is one of classic catatonic stupor, with marked improvement on continued hospital residence. It was possible to carry out a series of very careful physiologic studies upon this case, and the resultant findings indicate definite deviation in this field; these were particularly marked during the acute phase of the disorder and, in general, appeared to run directly parallel with essential fluctuations in the actual psychiatric status. In view of the attention now directed toward the physiologic substrate in schizophrenic disorder, it was felt that the presentation of such a study of a case which stood out clinically in such striking relief, would prove of interest; especially, since it emphasizes the paramount importance of what may be termed long-section study, in the proper evaluation of such material.

REPORT OF CASE

Clinical History.—F. W., a schoolboy, aged 14, was admitted to the State Psychopathic Hospital Sept. 6, 1921.

The father is an unsuccessful farmer. Both parents are extremely religious, and belonged to the "Crusader" sect prior to marriage; the home atmosphere was, therefore, intensely and narrowly religious. The mother is nervous, high strung and definitely of the zealot type. She has suffered several "breakdowns" which were characterized by irritability, restlessness and hypersthenic trends. The patient's father, who is thirteen years younger than his wife, is of very serious mind but is apparently not nearly so extreme in his views as she, although he had been a pastor in the "Crusader" church before he became a farmer. He gives a history of having had Pott's disease which, of late, seems to have become practically quiescent. For over a year, he has suffered from more or less intractable diarrhea, which is apparently associated with considerable pain and is considered by the family physician to be tuberculous in nature. The patient's older brother is reported to have suffered from tuberculosis of the hip, which is at present inactive. A maternal uncle is said to have experienced a "nervous breakdown" in his youth, while studying in a theological seminary. This continued for a number of months and was character-

*From the State Psychopathic Hospital, Ann Arbor, Mich.

ized by restlessness, ambivalence, and seclusiveness, with autistic trends. He was unable to continue his studies but undertook some evangelical work, in which he is still active. He has had several further "breakdowns" which were somewhat less severe but apparently of the same kind. The family history, as obtained, was otherwise negative.

The patient's birth was normal. He had the usual children's diseases and seems to have been particularly prone to bronchitis and tonsillar disorders. As a child he is described as frail, and very obedient; he never required punishment, was very religiously inclined, not gregarious and not especially fond of outdoor sports. He much preferred to remain at home reading and day-dreaming, a trend which was definitely fostered by the intensely religious and restrictive influences of his home. In school he was an exceptionally bright, assiduous pupil; in spite of interruptions, he completed the tenth grade at 14 and is described by his last teacher as of an extremely mature and receptive intellect.

For two years before admission the patient had been compelled, because of his father's failing health, to help considerably with the farm work. As a result of this he regressed somewhat physically. He had very little leisure time, turned from heavy farm work to study, and at times was forced, for varying periods, to interrupt his school attendance. These facts, added to the stress of attendant poverty seemed to depress and worry him, as he took his schooling with profound seriousness and was ambitious for a university career.

No frankly psychotic change was noted, however, until about a year before admission to the hospital when the patient was observed to have developed a mild, and at first rather undirected, nosophobia, which very soon became characterized by certain definitely psychopathologic trends. For example, he refused to open his mouth any more often or more widely than was absolutely necessary, but, when he did so, he was observed to expectorate vigorously afterward for some minutes. This reaction was found to be dependent upon a fear of admitting disease organisms into the mouth and causing a systemic infection. He became over-scrupulous in personal cleanliness, washed his hands almost incessantly and bathed much more frequently than usual. Following this, the patient began to complain of vague somatic distress, and later insisted that he had contracted appendicitis, in spite of the fact that repeated examinations by local physicians failed to reveal any evidence of such a process. He became progressively more seclusive, and in time seemed to lapse, almost wholly, into a state of autistic concern and reverie. He moved in a stereotyped fashion, assumed constrained postures, and finally became mute, refused to eat, and spent the entire day sitting or lying in stiff postures and staring into space. It is interesting to note that, at about the time of the onset of the mental change, the patient was observed by his father to have developed a definite bradycardia; the pulse rate rarely exceeded 60 and often fell as low as 48.

Clinical Course.—The patient's progress in the hospital was typically that of schizophrenic catatonia, with improvement after some five months of residence. On entry, he showed stereotypy of movement and posture, and lay all day prone upon his bed, with his hands close to his sides, and his gaze directed fixedly to the ceiling; yet he was, apparently, fully alive to the general environmental situation. Whenever he left his bed, he did so in a slow, inannikin-like fashion and, upon returning, went through a certain set, stereotyped routine, particularly as to tucking the bed-clothes under his armpits. He was absolutely mute except in the morning and evening when he would kneel by the

bed and pray in a whisper. He was further negativistic in that he refused to eat or even to open his mouth, and it was necessary to feed him by tube for almost five months. He appeared completely indifferent to painful stimuli and to threats; in short, he presented a typical picture of acute stuporous catatonia.

Examination.—During this phase, except for some degree of general emaciation and asthenia, the patient showed no gross somatic deviation, though there were certain features indicative of the carnivorous body type, or habitus, of Bryant.¹ Of late this has come to be construed as a frequent associate of the clinical vagatonias of Eppinger and Hess. The characteristics were: flat thorax; somewhat long, thin neck; long tapering fingers; cyanotic extremities; low tension pulse, with bradycardia (46 to 58 per minute); hypopiesia (systolic 90 to 100, diastolic 65 to 85); coarse, dry, rather darkly pigmented skin; winged scapulas and visceroptosis. The axillary temperature range during this period was from 96 to 97 F. Roentgenologic study of the skull, thorax and extremities yielded no evidence of endocrinopathic or other pathologic deviation, and examination of the urine and blood were entirely negative.

Neurologic examination disclosed nothing of note except mydriasis and marked dilator dermatographia; the latter at times took on a highly specific and localized character; thus, frequently, particularly after physical pain or affective stress, one hand became red, engorged, and actually hot, while the other appeared cyanosed, frigid, and of distinctly lesser size.

Pharmacodynamic studies afforded frank evidence of a vagotonic trend. Pilocarpin, one-eighteenth grain (0.004 gm.), induced very marked sudomotor response; one-quarter milligram of physostigmin caused cardiac slowing, and one-hundredth grain (0.0006 gm.) of atropin produced but slight tachycardia. The oculocardiac reflex, however, was negative. One milligram of epinephrin, given subcutaneously, was practically without effect; the pulse acceleration was consistently less than 10 and the rise of blood pressure was only 2 to 5 mm. The increase in basal metabolic rate which followed the intramuscular administration of 1 mg. of epinephrin, was only +7 per cent. (average normal +20 per cent.), and the glycemic response over a period of six and one-half hours, was characterized by definite lowering of the acme point, very gradual decline, and the absence of any indication of a normal reaction pit or subsequent rebound. These results correspond essentially with those found recently in other cases of acute schizophrenic disorder, by Parsons and Raphael.² Negative responses were obtained with the Löwi, Csepai, and Ascoli³ tests for adrenal function and also with the Ascoli and Brock-Kay⁴ tests for hypophyseal function.

The blood platelet count was somewhat elevated when compared with the average normal (505,000 per cubic millimeter), and the bleeding time was decreased four minutes. Blood sugar tolerance (Janney-Isaacson schedule⁴ and

1. Bryant, J.: *The Carnivorous and Herbivorous Types in Man*, Boston M. & S. J. **172**:321 (March 4) 1915.

2. Raphael, T., and Parsons, J. P.: *Metabolism Studies in Dementia Praecox and Manic Depressive Insanity*, Arch. Neurol. & Psychiat. **8**:172 (Aug.) 1922.

3. Rabinowitch, I. M.: *Metabolic Studies on a Case of Diabetes Insipidus*, Arch. Int. Med. **28**:355 (Sept.) 1921.

4. Janney, N. W., and Isaacson, V. I.: *A Blood Sugar Tolerance Test*, J. A. M. A. **70**:1131 (April 20) 1918.

standard Folin technic) was definitely delayed and there was evidence of initial hypoglycemia, as had been noted also in other acute schizophrenic cases.⁵ The figures were: 0.07 per cent. at the fasting level; 0.14 per cent. at the end of the first hour; 0.16 per cent. at the end of the second hour, and 0.11 per cent. at the end of the third hour.

The basal metabolic rate was consistently low, and ranged from -21 per cent. to -29 per cent., in spite of a tube-fed diet which contained 3,200 calories in twenty-four hours; definite anoxemia (Van Slyke) was noted both in the arterial (-15 per cent.) and in the venous (-30 per cent.) blood, although on the basis of the hemoglobin content this did not seem to be associated with deficiency in oxygen carrying power. Blood fragility⁶ was found to be slightly, but consistently, increased, and the blood lipid content (Hoag modification of the Bloor technic⁷) was somewhat diminished (0.5 per cent.). Hepatic function⁸ was found to be definitely delayed; the phenoltetrachlorophthalein return time was thirty-four minutes as compared to the average normal interval of seventeen minutes.

Later Course and Examinations.—After about five months in the hospital, during which the patient had become profoundly emaciated, a gradual tendency toward improvement became evident. He began to eat and showed some affective response. From this time, a gradual increase in weight was noted until at the time of discharge, nine months after admission, the patient had gained 50 pounds. Mentally, improvement was almost as marked. The patient began to talk, played games, drew, painted, mingled with the other patients, and joined in the outdoor games. Yet this improvement seemed distinctly incomplete as he still occasionally showed mannerisms in gait, and was somewhat seclusive in reaction; there was continued evidence of intrapsychic conflict, manifested by persistent refusal to answer questions which concerned intimately personal problems, and by indications of a certain sullenness and dysaffect when such questions were asked. Nevertheless, it was very evident that vast improvement had actually taken place and, paralleling this, it was observed that the disturbances in the endocrino-autonomic and metabolic fields were also gradually disappearing. Particularly was this true of the vagotonia. A physiologic cross-section, completed a few weeks prior to discharge, indicated that practically normal responses were now obtained in all fields which had previously shown deviations.

The basal metabolism was now normal (+5 per cent.), as were the blood lipid content (0.69 per cent.), hepatic function (19 minutes), arterial and venous blood-oxygen combining power, blood fragility, blood sugar tolerance except for an initial relative hypoglycemia (0.08, 0.12, 0.08, 0.07), blood platelet count (270,000 per cubic millimeter) and bleeding time (5.4 minutes).

5. Raphael, T., and Parsons, J. P.: The Physiologic Level in Dementia Praecox, *Am. J. Psychiat.*, to be published.

6. Greenthal, R. M., and O'Donnell, W. J.: Fragility of Red Blood Corpuscles in Infectious Diseases, *Am. J. Dis. Child.* **22**:212 (Aug.) 1921.

7. Hoag, L. O.: Personal Communication, University Hospital, Ann Arbor, Mich.

8. Aaron, A. H., Beck, E. C., and Schneider, H. C.: The Phenoltetrachlorophthalein Test for Liver Function, *J. A. M. A.* **77**:1631 (Nov. 19) 1921.

Responses to the Löwi, Csepai, Ascoli, and Brock-Kay tests were likewise normal. The reaction to autonomic pharmacodynamic reagents showed complete absence of vagotonia. In fact, there seemed to have developed a slight sympathetic preponderance, as the pilocarpin and physostigmin tests were frankly negative and atropin produced definite tachycardia (+50); epinephrin produced pronounced pallor, tremor, pulse acceleration (+65), blood pressure rise (+25) and glycosuria. At this time, too, it is interesting to note that the blood pressure had become definitely elevated as compared with the level on admission (systolic 120, diastolic 70). The body temperature was now quite normal (98 to 98.6 in the axilla), as was the vasomotor status except for a mild, persistent tendency to cyanosis of the extremities. There was no longer any evidence of hyperpigmentation.

DISCUSSION

In view of the familial setting and the apparently classical pathogenetic and clinical course, a diagnosis of acute catatonic stupor, of schizophrenic type, with subsequent remission, seems warranted. It is also permissible to assume the existence of a primary asthenic (carnivorous) habitus from the somatomorphologic deviations with associated vagotonic cardiovascular features which have been described.

The existence of a fundamental autonomic imbalance, which was at first definitely of the vagotonic type, and later, as recovery progressed, assumed that of a mild sympathicotonia, may be inferred during the first phase from: the bradycardia; hypopiesis; cyanosis; striking vasomotor disorder; hypothermia; coarse, thick pigmented skin; strongly positive pilocarpin and physostigmin reactions; lowered epinephrin and atropin sensitivity; and the generally depressed metabolic status. During the second phase imbalance may be inferred from: the mild tachycardia; slightly hyperpietic trend; improvement in the hypothermia, skin pigmentation and vasomotor disorder; and from the definite pilocarpin and physostigmin insensitivity, with markedly increased sensitivity to epinephrin and atropin. The significance of this reversal is admittedly more or less conjectural; but it obviously seems to be of the nature, either of a primary autonomic compensation, or a reflection of improved adrenal function. The actual development of such a clearly cut vagotonia upon a primarily asthenic basis may be construed as a corroboration of the assumption that a certain relationship exists between this habitus and clinical vagotonia.

That there seems also to have been a definitely endocrinic facet to the clinical structure, is obvious. During the preliminary or acute period, there was adrenal hypofunction accompanied, though to a somewhat less degree, by pituitary and thyroid inadequacy. During the recovery phase, there was gradual improvement in this state of hypofunction and, eventually, the development of a slight but definite hyperactivity

of the adrenal system. During the stupor, adrenal hypofunction was evidenced by the occurrence of the well marked vagotonic features⁹ which have been described. As evidence of relative thyroid hypofunction, there may be noted: the general asthenic reaction and stupor; the coarse skin texture; bradycardia; low body temperature; low blood pressure; low blood sugar; diminished basal metabolism; relative insensibility to epinephrin; increased blood platelet count and shortened bleeding time. As evidence of diminished hypophyseal activity, there were the general asthenia; the cardiovascular disturbances; and the hypothermia, metabolic depression and negative response to the Ascoli and Brock-Kay tests. During the convalescent phase the change which had occurred is indicated by the practically normal pharmacodynamic and endocrinic responses; the wholly normal responses in the metabolic field; the marked general physical improvement; and the disappearance of the initial vagotonia with the gradual development of frankly sympathicotonic features.

The actual significance of this phasic alteration of endocrine function, especially in the autonomic field, is more or less speculative; but presumably it is associative rather than specifically primary or secondary in nature. At any rate, on this basis there appears to exist an intimate interrelationship between this endocrinic triad and the sympathetic and parasympathetic divisions of the autonomic nervous system. This is in accord with the observations of Brown,¹⁰ Timme⁹ and Cannon.¹¹

As evidence of the general metabolic depression, essentially hypoxidative in type, which occurred during the acute stuporous period, there are: the consistently lowered basal response in spite of high caloric feeding; delayed sugar tolerance with initial hypoglycemia; relative hypolipemia; diminished blood-oxygen combining power; increased red cell fragility; and relative hepatic hypofunction; as well as the development of a generally asthenic state; depression of the cardiovascular field; and indications of lessened function of the adrenals, thyroid and pituitary. These findings, it may be of interest to note, appear to be in close agreement with determinations upon other schizophrenic subjects by Raphael and Parsons.⁵ The question of the specific significance of the metabolic deviation is obviously also conjectural, particularly as to whether it is primary, or secondary to the endocrino-autonomic disorder.

9. Timme, W.: *Clinical Endocrinology*, Neurol. Bull. **3**:3 (Jan.) 1921.

10. Brown, L.: *The Sympathetic Nervous System in Disease*, London, 1920, p. 23.

11. Cannon, W. B.: *Evidence of Nervous Control of Some Internal Secretions*, Arch. Neurol. & Psychiat. **6**:707 (Dec.) 1921.

SUMMARY

An analytic report is made of a case which presented definite indications of primary or fundamental asthenic habitus, the gradual development of an acute schizophrenic catatonia with stupor reaction, and almost complete recovery; and which further, in strikingly phasic association, on the physiologic level, presented marked vagotonia and hypoadrenalism, and, to a somewhat less degree, hypophyseal and thyroid inadequacy with evidence of general metabolic depression, essentially hypo-oxidative in type. These findings strongly emphasize trends that have been noted in other acute schizophrenic reactions.

PHENOBARBITAL (LUMINAL) TREATMENT OF INSANE EPILEPTICS

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Phenobarbital (phenyl-ethyl-barbituric acid), under the name of luminal, was patented in the United States during 1912. It was accepted by the Council on Pharmacy and Chemistry of the American Medical Association and incorporated in New and Nonofficial Remedies during 1913. In this book it was reported as a useful hypnotic in nervous insomnia and conditions of excitement of the nervous system. No mention was made of its possible utility in the treatment of epilepsy.

As early as 1912, Hauptmann¹ reported tentatively favorable results from the use of this drug in treating a series of cases of idiopathic epilepsy. In 1913 Pécheux² had good result in ten cases of epilepsy. After this, numerous reports appeared in the foreign literature, all quite favorable in character. Fuchs,³ Debrowski⁴ and Kutzinski⁵ were among those who wrote hopefully about the new drug. In 1919 Dercum⁶ reported exceptional success in cases of mild idiopathic epilepsy. Grinker,⁷ in 1920, reported favorable results in a series of one hundred cases treated by him since 1913. In this paper he calls attention to his reference to the use of this drug in discussing a paper read by Dercum before the Section on Nervous and Mental Diseases of the American Medical Association in 1916. During 1920 Cheinisse⁸ reviewed the foreign literature and reported that the consensus of opinion appeared to be that this drug deserves the leading place in the treatment of idiopathic epilepsy. In 1921 Kirk⁹ reported favorable results in a series of two hundred cases. Austin¹⁰ reported forty-nine cases treated for a period of fifteen months, and was of the opinion that phenobarbital gave better results in the treatment of idiopathic epilepsy than any other drug so far used. Small¹¹ reported fifty cases, 80 per cent of which

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1. Hauptmann, A.: Münch. med. Wchnschr. **59**:1907 (Aug. 27) 1912.
 2. Pécheux, Alfred: Lille, 1913, No. 25, p. 100.
 3. Fuchs, W.: München. med. Wchnschr. **61**:873 (April) 1914.
 4. Debrowski, W. G.: Monatschr. f. Psychiat. u. Neurol. **36**:248, 1914.
 5. Kutzinski, A.: Monatschr. f. Psychiat. u. Neurol. **36**:174, 1914.
 6. Dercum, F. X.: Therap. Gaz. **35**:609, 1919; J. Nerv. & Ment. Dis. **50**:168 (Aug.) 1919.
 7. Grinker, Julius: J. A. M. A. **75**:588 (Aug. 28) 1920.
 8. Cheinisse, L.: Presse méd. **28**:598 (Aug. 28) 1920.
 9. Kirk, C. C.: Am. J. Insan. **77**:559 (April) 1921.
 10. Austin, M. L.: Ohio State M. J. **17**:683 (Oct.) 1921.
 11. Small, V. R.: Virginia M. Month. **48**:386 (Oct.) 1921.

improved under treatment. Hughes¹² reported thirty cases treated for one month, with reduction in number and severity of the seizures. Sands¹³ treated sixty-eight insane, idiopathic epileptics with very satisfactory results. Foley¹⁴ reported the result of treatment in fifteen men and twenty women who were under institutional care because of epilepsy. He stated that there was no reduction in the number of seizures during the three months in which the men were given phenobarbital instead of bromid; but that among the women there was a reduction from two hundred and five seizures in thirty days during the last month under bromid, to sixty-six seizures during the third month under phenobarbital. This prominent difference in the effect of the drug, drawn upon sex lines, must be carefully checked by other observations before being given serious credit. Foley further stated that "the severity of each convulsive attack of those having convulsions, was greatly increased. . . . we noticed a marked increase in the irritability of all cases under treatment and the irritability of some cases increased to a deep furor. The furors were more prolonged and intense than ever observed under the bromid treatment." Golla¹⁵ treated one hundred and twenty-five patients with phenobarbital. Eighty-nine of these did better than when given bromid, four persisted in complaining of giddiness so that the drug had to be discontinued, two had urticarial rashes during early treatment that disappeared without the drug being discontinued. Fox¹⁶ treated sixteen children and adolescents with combined phenobarbital and bromid, with varied effects on the intellectual condition of the patients. On the whole, his impression as to the value of the drug appeared quite favorable.

In 1912 and 1913 German physicians, using phenobarbital for its sedative effects, reported toxic action and rashes after even moderate doses. Blichert,¹⁷ in July, 1913, reported signs of intoxication in five out of thirteen patients who were taking from six to nine grains (0.4 to 0.6 gm.) of phenobarbital twice daily. In addition to the intoxication, several of his patients developed transient rashes. Farnell¹⁸ reported two patients with severe toxic symptoms after taking nine grains (0.6 gm.) and eighteen grains (1.2 gm.) respectively. These patients are described as "dreamy and sleepy . . . speech was slurring and scanning; . . . on attempting to walk she

12. Hughes, W. N.: *Rhode Island M. J.* **4**:157 (Dec.) 1921.

13. Sands, I. J.: *Arch. Neurol. & Psychiat.* **5**:305 (March) 1921.

14. Foley, E. A.: *Illinois Instit. Quart.*, March 31 and June 30, 1921.

15. Golla, F.: *Brit. M. J.* **2**:320 (Aug. 27) 1921.

16. Fox, J. T.: *Lancet* **2**:558 (Sept. 10) 1921.

17. Blichert, E.: *Ugesk. f. Læger* **75**:1149 (July 3) 1913.

18. Farnell, F. J.: *J. A. M. A.* **61**:192 (July 19) 1913.

swayed." Both of these patients recovered within forty-eight hours. Haug¹⁹ reported two patients, each taking a grain and a half (0.09 gm.) of phenobarbital three times a day, who developed toxic symptoms eleven days and four weeks, respectively, after starting to use the drug. These patients had high fever, diarrhea, mucous stools, (blood in the stools of one) and a scarlatiniform eruption over the entire body except the hands and face. Both patients recovered a few days after the suspension of the drug. Ruggles²⁰ reported toxic effect from a dose of three grains (0.2 gm.) daily. His patient showed slowing and slurring of speech, incoordination, tremor of the lips, and lack of facial expression. Recovery took place within a few weeks. Rosenberger²¹ reported a death following the use of phenobarbital and opium combined. There have been many other reports on the treatment of epilepsy with phenobarbital and the toxic action of the drug, but those quoted are typical of the whole and are sufficient to indicate the trend of opinion as to its efficacy and the dangers of indiscriminate, ill advised use of the drug.

It is the purpose of the present report to show the results of treatment with phenobarbital in a series of male patients at the Warren State Hospital for the Insane. The patients are all classed as insane epileptics and require hospital care because of pronounced dementia or periods of extreme and dangerous violence. None of the series can be considered of the mild or incipient type, and nearly all have been subject to seizures for many years. All of the patients treated are considered as belonging to one of four groups: idiopathic, traumatic, senile, or syphilitic. For the purpose of this report epilepsy will be considered as meaning any illness in which the most prominent symptom is clonic convulsive states associated with unconsciousness. When epilepsy is modified by the word "idiopathic" it will be understood that careful physical, neurologic, mental, and laboratory examinations have failed to reveal any probable cause for the convulsive attacks. Traumatic epilepsy is diagnosed only when definite evidence of cranial injury can be demonstrated and associated with the development of the seizures. Senile epilepsy is diagnosed when the seizures develop well after middle life, and when there are other clear signs of cerebral sclerosis. Epilepsy is considered syphilitic when there is, or has been, laboratory evidence of syphilis during examination of the spinal fluid.

During January, 1920, following the earnest solicitation of Dr. Dercum, phenobarbital was tried in the treatment of fifteen hospitalized, insane epileptics of the idiopathic type. The initial results were

19. Haug, W.: München. med. Wehnschr. **66**:1494 (Dec. 26) 1919.

20. Ruggles, A. H.: Arch. Neurol. & Psychiat. **6**:234 (Aug.) 1921.

21. Rosenberger, F.: Med. Klin. **15**:1150, 1919.

favorable enough to interest me in continuing the treatment and adding others to the phenobarbital group. In the discussion of a paper by Kirk,⁹ read before the 1920 meeting of the American Psychiatric Association, Dr. H. W. Mitchell presented the results obtained in this preliminary group of cases. He said, "The most noticeable effect upon the patients under observation was the improvement in their daily reactions to ward life. There was much less irritability and consequent quarreling, a great reduction in the minor injuries sustained during seizures, and no exhibition of the periods of clouded consciousness which the same patients had manifested frequently before the use of the drug. So far as could be detected, there were no noticeable bad effects from the use of the drug." The effect upon the number of



Chart 1.—Number of seizures in fifteen men during eight months under treatment with: C, no drugs; P, phenobarbital; P B, phenobarbital combined with bromids.

seizures is indicated in Chart 1. The dots represent the average number of seizures for the entire group of fifteen patients during each of the eight months covered by the study. As will be seen, the first four months were used as a control period during which no drug treatment was employed. In the fifth month a combination of phenobarbital and bromid was given, and a remarkable reduction in the average number of seizures followed. During the sixth month phenobarbital alone was used, and there was a moderate increase in the number of attacks. During the seventh month all drugs were omitted and the number of seizures increased a little more. During the eighth month when phenobarbital alone was used, there was a reduction in the number of seizures.

Following this initial trial of phenobarbital, a standardized technic of administration was developed. Every morning, before breakfast, a saline laxative is given. The type of laxative has been changed from time to time and the dosage varied according to the requirements of the individual patient. Midway between the noon and evening meals one and one-half grains (0.09 gm.) of phenobarbital are administered, preferably in capsule. The phenobarbital is given only five days in each week. For convenience and clearness, the nurses' instructions read that it is to be given every day except Saturday and Sunday of each week. The diet is regulated along the lines recommended by Spratling.²² Regularity of time, moderation in quantity, full mastication, and limitation in amount of the richly nitrogenous foods are considered the most important requirements. Some patients require a larger amount of phenobarbital to obtain the best results and such increases are made when indicated. Divided doses have been tried; but a single dose, given in the afternoon when the stomach is empty, is thought to be the best mode of administration. As much as four and one-half grains (0.25 gm.), five days in each week, have been given for several weeks without ill effects; but three grains (0.2 gm.), given five days in each week, have caused mental dullness and a slight rash. No toxic effect has been observed from the use of a grain and a half (0.09 gm.) administered in this manner. Whenever the larger doses are used it is essential that the nurse or the family be carefully instructed concerning the toxic symptoms, and that the physician be informed immediately if any of them are even suspected. Further, it is necessary for the physician to keep constantly in mind that he is prescribing a powerful drug, and to insist upon frequent, personal observation of his patient, especially if more than a minimum dose is used.

Beginning Feb. 1, 1921, phenobarbital was given to thirteen men with idiopathic epilepsy. Nov. 1, 1922, twelve of these patients were still in the hospital and receiving the same type of treatment. The regimen during the entire period was the same each month except during February, April, and July of 1922. During February twenty grains (1.25 gm.) of mixed bromids daily were substituted for the phenobarbital with the purpose of observing the effect of change in treatment. In April the same change was made because there was difficulty in obtaining phenobarbital. In July phenobarbital was given to some patients and mixed bromids to others. One patient died during April in status epilepticus. This man averaged seventeen seizures per month for years prior to taking phenobarbital and had developed status

22. Spratling, W. P.: *Epilepsy and Its Treatment*, 1904, W. B. Saunders Company, p. 354.

epilepticus several times. In January, 1921, while taking phenobarbital, a similar condition developed; and in April, 1922, while taking mixed bromids, the final attack occurred. The ten months which elapsed between March, 1921, and February, 1922, during which he was taking phenobarbital, were the most comfortable he had had for years.

An attempt has been made to show in graphic form the average number of seizures of this group during each of the months covered by this report, and to compare these numbers with the monthly average for the group when no specific drug treatment was used. In making this graph a careful estimate of the average number of attacks per month for each patient prior to taking phenobarbital was made by consulting hospital records, or by interviews with members of the man's family. A period of at least three months was considered in each instance. The average number of seizures per month for each of the thirteen men were added together and the result was divided by thirteen. This gave eight seizures per month as the average, or norm, for this particular group; and it is indicated by the straight line in the graph. The number of seizures of each patient, during each month was then added, divided by thirteen, and this result compared with the norm for the group. Nine of this group had a lower average number of seizures per month while being given phenobarbital, three showed no definite change, and one had more seizures than before.

The second group is very similar to the first, the main difference being that the patients have been treated only from eight to thirteen months. It is made up of twelve men, seven, who have been in the hospital for a number of years, and five who have been recently admitted. Before treatment was started this group averaged nine seizures per month. Chart 3 compares this norm with the group averages for each of the months covered by this report. Changes from the routine treatment were made during the months of February, April, and July, as described in the explanation of Chart 2. Eleven of these men had a marked reduction in the number of seizures while taking treatment and one continued to have about his usual number of attacks. As will be seen by consulting the graph, there was a quite distinct rise in the average number of seizures during the months that bromid was substituted for phenobarbital. Six of these patients had a distinctly smaller number of seizures when taking phenobarbital than when taking bromid. The other six appear to have done equally well with either form of treatment.

The next group is made up of men who have developed grand mal epileptic attacks following cerebral trauma. None of the injuries is recent, and all of the patients have had the benefit of operation, or at

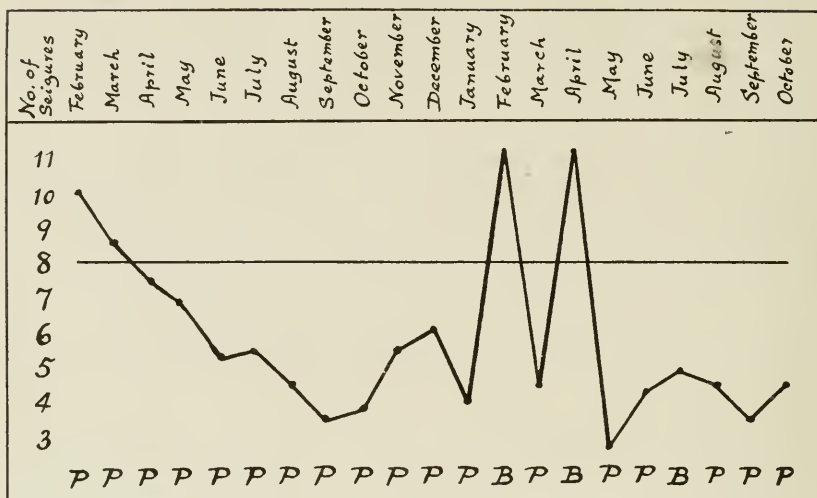


Chart 2.—Average number of seizures in thirteen men under treatment with: *P*, phenobarbital; *B*, mixed bromids. The horizontal line represents the previous average. The same symbols are used in Charts 3, 4, 5 and 6.

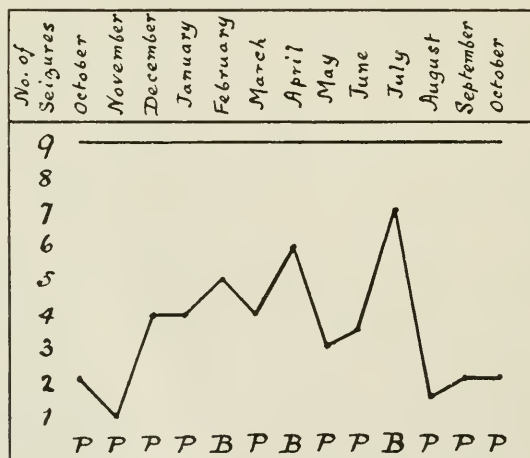


Chart 3.—Average number of seizures in twelve men.

least of competent surgical advice. Several have submitted to three or four operations. The average number of seizures for this group before taking treatment was found to be eleven per month. Treatment with phenobarbital was started Oct. 1, 1921, and has been continued since that time, except for three months—February, April, and July of 1922. During these months the treatment was the same as described in the explanation of Chart 2. The chart shows that there has been a great reduction in the monthly average of attacks. During the months that bromid was substituted there was some rise in the number of seizures, but no pronounced increase. Two of the patients had many

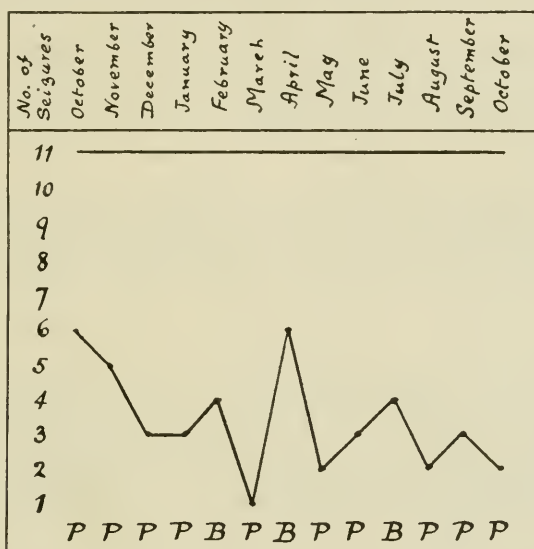


Chart 4.—Treatment of six men with grand mal seizures after cerebral traumatism.

more attacks during the months that bromid was used, the other four did about equally well with either type of treatment.

The fourth group is made up of six cases diagnosed senile epilepsy, that have been treated for from eight to thirteen months. The results of treatment in the individual cases of this type were not so good as the graph indicates. One of the old men averaged ten seizures a month without treatment; but, while taking phenobarbital or bromid, has had only four seizures a month. The other five had from one to five seizures a month prior to treatment, and they have all continued to have about the same number since. Chart 5 compares the monthly averages of this group with the group norm in the same manner as the preceding charts.

Two patients have been treated who have typical grand mal attacks associated with the usual type of epileptic mental deterioration, but who have had, at some time during their period of hospital care, positive blood Wassermann reactions and syphilitic colloidal gold curves. Neither of these men has had positive Wassermann reactions with the spinal fluid, nor do they have the neurologic signs ordinarily present in general paralysis or cerebrospinal syphilis. Antisyphilitic

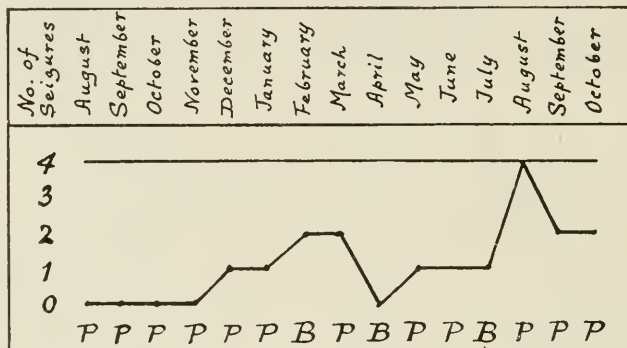


Chart 5.—Treatment of six cases of senile epilepsy.

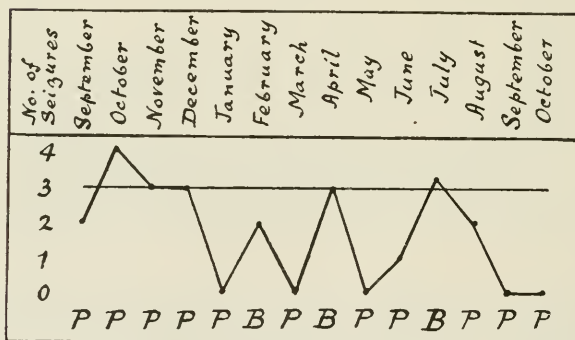


Chart 6.—Treatment of two epileptics who have given positive Wassermann tests at some time.

treatment has been tried with little, if any, effect upon the number of seizures, though there has been improvement in the laboratory findings. Chart 6 compares the average number of attacks per month of this group with the group norm, in the same manner as the preceding charts. There appears to be some reduction in the number of seizures, but the frequency of the attacks is so irregular and the group so small that little information can be obtained from the data at hand.

SUMMARY

1. Phenobarbital has a cumulative effect that appears to be successfully combatted by a break of two days in each week during its administration.

2. A sudden break in the administration of phenobarbital is sometimes followed by a series of seizures. There is much less danger of such trouble if bromid is given as soon as the phenobarbital is withdrawn.

3. One and one-half grains (0.09 gm.) given five days in each week appears to be a safe dosage. If larger amounts are given, very careful observation is necessary to detect possible toxic symptoms early and to prevent the more serious disorders.

4. The use of this drug may be followed by: rash, simulating measles or scarlet fever; symptoms like those of alcoholic intoxication; severe, cholera-like diarrheas; mental hebetude; delirious states; and other like troubles.

5. Favorable results were obtained from the administration of phenobarbital in cases diagnosed as idiopathic and traumatic epilepsy. The results in cases diagnosed as senile and syphilitic epilepsy were doubtful.

6. Phenobarbital is not to be considered as a "cure" or specific for epilepsy.

7. Phenobarbital and bromid may be combined and better results thus obtained in selected cases.

8. Each case should be considered as an individual problem and phenobarbital, bromid, or a combination of the two used according to the results.

THE PATHOGENESIS OF EPILEPSY

A CRITICAL REVIEW WITH A NEW INTERPRETATION
OF THE AVAILABLE DATA *

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NEW YORK

The conceptions of the causes of epilepsy are many and confusing. Any discovery which reveals the causes underlying the convulsive manifestations will tend to clarify the situation. I have little sympathy with the view so often expressed of late that the mental (dementia), and even the paralytic and other somatic disorders of epilepsy, are secondary to the psychogenically determined convulsive attacks. The more I study the experimental work and the anatomic, biochemical and pathologic data bearing on the question, the more tenaciously do I hold to the belief that if the convulsions in epilepsy are once satisfactorily explained, the whole problem will be placed on a sound basis.

While it is not possible to synthesize all theories of the causation of epilepsy, the psychogenic factor may be readily assimilated in our discussion. The staunchest upholder of the psychoanalytic conception is Dr. L. Pierce Clark.¹ He believes that epilepsy is the result of a peculiar personality defect which is constitutional. This defective make-up is characterized by great egocentricity, emotional poverty and hypersensitiveness. Thus, the epileptic is handicapped in his ability to make even fundamental life adaptations and adjustments. To the stresses which constantly accumulate, he reacts by convulsive seizures. This allows him to escape from reality and responsibility and aids him to protect his personality from the demand to cope with his environment. According to Clark, therefore, treatment cannot be successful until the approach is made by psychobiologic, analytical means. In evaluating the psychogenic, emotional factor, I venture to quote remarks I made when Dr. Clark's last paper on this subject was read at the June, 1922, meeting of the New York Neurological Society.²

* Read at meetings of the Clinical Society of the Post-Graduate Hospital, Oct. 6, 1922, and of the New York Neurological Society, Nov. 14, 1922.

1. Clark, L. P.: A Psychological Interpretation of Essential Epilepsy, *Brain* **43**:38 (May) 1920; Treatment of the Epileptic, Based on a Study of the Fundamental Makeup, *J. A. M. A.* **70**:357 (Feb. 9) 1918; Clinical Studies in Epilepsy, *Psychiat. Bull.*, January, 1916, April, 1916, and January, 1917; Some Suggestions for More Accurate Mental Therapy in Epilepsy, *J. A. M. A.* **71**:255 (July 27) 1918.

2. Clark, L. P.: *Arch. Neurol & Psychiat.* **8**:319 (Sept.) 1922.

"I believe that Dr. Clark stated that the delirium in petit mal was of the functional type because its content dealt with the everyday life experience of the individual. I do not think that is any criterion. All delirial productions deal with ordinary life experiences, with what is in the consciousness. Therefore, this does not prove that the delirium in petit mal is functional and not organic. I think there is danger in laying too much stress on the makeup and personality as determining the mental disease from which the patient suffers. The three points which Dr. Clark emphasizes—egocentricity, emotional poverty and hypersensitiveness—are not different from those found in the personality of patients with dementia praecox. The type of reaction is influenced by the makeup, but this does not cause the disease itself. Dr. Clark said that a tyro can pick out the epilepsy makeup, but that the epileptic patient may have minor degrees of the same attributes and that only the carefully trained physician can bring these out. The same qualities that are supposed to belong to the epileptic makeup are found in the characters of many people. Why should the mechanism be different in epilepsy if it is of psychogenic origin? Why should the psychologic mechanism work differently in epilepsy and express itself in convulsive manifestations which are objectively and by various chemical and other means so readily differentiated from the convulsive manifestations of hysteria?"

The psychogenic theory of the causation of epilepsy does not explain why other nonepileptic individuals who are egocentric, emotionally defective and hypersensitive do not have the convulsive episodes of the epileptic. Neither does it explain the epilepsies of cerebral arteriosclerosis, trauma, the Jacksonian attacks, or the convulsions occurring in general paralysis, cardiorenal disease, endocrine disturbances (possibly pituitary, status thymico-lymphaticus), and the focal epilepsies of brain tumor, brain abscess or the epilepsies in animals (monkey).³ In all fairness, however, these psychologic explanations of the epileptic constitution, cannot be entirely disregarded. It requires only the barest reference to the work of Crile⁴ and Cannon,⁵ to deter one from adopting such an attitude. These authors have shown that painful and prolonged emotional states cause increase of the adrenal secretion in the blood, and, with the addition of fatigue, result in toxic changes which are destructive to liver and brain cells. The blood in these conditions also manifests increased coagulability and sugar content. In view of the work on the toxicity of the blood in epilepsy, to be discussed later, one cannot too hastily disregard this situation in its possible relation to the convulsive manifestations. The liver is now known to produce a pressor and cardio-accelerator protein⁵ in response

3. Sherrington, C. S.: Stimulation of the Motor Cortex in a Monkey Subject to Epileptiform Seizures, *Brain* **41**:48 (May) 1919.

4. Crile, G. W.: *Boston M. & S. J.* **153**:893, 1910; also many other more recent publications in *The Journal A. M. A.*, 1910-1921.

5. Cannon, W. B.: *Bodily Changes in Pain, Hunger, Fear and Rage*, New York, D. Appleton & Co., 1920, p. 187. *New Evidence for Sympathetic Control of Some Internal Secretions*, *Am. J. Psychiat.* **2**:15 (July) 1922.

to sympathetic stimulation (hepatic nerves). Insufficient and inefficient personalities would yield more promptly to the disturbing emotional factors which produce these phenomena than more stable individuals.

The mechanical and vascular theories of the pathogenesis of the convulsions in epilepsy are satisfactorily summarized by Percy Sargent.⁶ Most of our knowledge concerning this phase of the subject is derived from work on laboratory animals by Russell,⁷ Sherrington and others, and on human beings by various surgeons including Sargent, Krause and Cushing.

The most reliable data point toward the vascular system, either alone or as a carrier of toxic products, as the offending element in epilepsy. Some believe with Sargent⁶ that the vascular disorders are due to disturbance of the sympathetic-autonomic control of the cerebral cortical vessels, while others incriminate the endocrine or other biochemical intoxicants carried to the cortex in the blood stream, as the primary factor in the causation of the convulsive seizure. These toxic substances may be manufactured locally in the brain by the break-down of cortical tissue in disease (arteriosclerosis, general paralysis, uremia, the convulsions of alcoholism and of status thymico-lymphaticus, etc.).

Sargent noted "the striking rapidity with which visible circulatory changes may occur in response to various disturbing causes." It was not the congestion or anemia of the brain as a whole, but the local variations and abnormalities observed in cases of trauma and brain tumor with epileptic manifestations, which made him certain that "if there is a fundamental precipitating cause for all convulsive attacks, any hypothesis which does not take into account the pathogenesis of fits originating in the neighborhood of a gross lesion, must fail to touch that essential point."⁶

Sargent agrees with Russell, who first drew an analogy between the experimental animal in a condition of decerebrate rigidity and the tonic spasms of the epileptic in the initial tonic, rigid phase of the convulsive seizure; and, for the moment, the unconscious rigid epileptic is comparable to the animal whose cerebral circulation has been shut off. Of course, this does not account for the clonic movements which may follow. In laboratory animals, as many have observed, when the cerebral circulation is cut off, clonic convulsive movements supervene.^{8,9}

6. Sargent, Percy: Some Observations on Epilepsy, *Brain* **44**:312 (Nov.) 1921.

7. Russell, A. E.: Goulstonian Lectures, London, 1909.

8. Starling: Human Physiology, Philadelphia, Lea & Febiger, pp. 984, 989, 990, 1002, 1003 and 1085.

9. Stewart, G. N.; Guthrie, C. C.; Burns, R. L., and Pike, F. H.: The Resuscitation of the Central Nervous System of Mammals, *J. Exper. Med.* **8**:289, 1906.

Pike,⁹ ten years ago, shut off the cerebral circulation of cats by tying off the internal carotid and vertebral arteries close to the subclavian. Immediate unconsciousness and paralysis of respiration (asphyxia) occurred; to prevent death of the animal, artificial respiration was kept up for the eight or ten minutes during which the occlusion was allowed to continue. At the end of this time, the arterial sutures were loosened and the circulation was resumed. Immediately the animals were thrown into a series of convulsions, which lasted for days. Any jarring, stroking of the fur, or even loud noises, were sufficient to cause typical convulsions, a tendency which at times lasted for weeks.

Without developing the idea further at this point, it must be remembered that cells cut off from their blood supply even momentarily show a change in reaction toward acidity. During asphyxia and violent exercise, lactic acid is formed in the muscles and appears in the blood and urine. Cuneo has shown that, in acid mediums, the normal nucleohistone content of brain and other cells, (leukocytes) is capable of breaking down into nucleinic acid and proteoses (albumoses). These latter substances are capable of causing convulsions, whether they are painted on the cortex or are injected intravenously. In connection with the experiment of Pike, it has been noted in a few cases of human epilepsy that the tonic phase of the fit occurred coincidentally with arrest of the circulation, while the clonic phase was ushered in with the resumption of the radial pulse and the heart beat.⁶

The clonic movements which follow the tonic spasm, have the same origin as the motor mechanism which causes the Jacksonian fit. These Jacksonian movements are exactly like those produced by experimental faradization of the motor cortex and are not preceded either by the tonic phase or by loss of consciousness. This is the chief objection to the theory that the epileptic convulsive attack is caused in its entirety by the vascular disturbances described by Sargent in his cases of traumatic epilepsy. Sargent and others have repeatedly called attention to the fact that unconsciousness is the first sign of occlusion of the cerebral circulation. The theory is advanced that the clonic movements are caused by the gradual and, at first, insufficient resumption of the blood supply in the constricted cortical vessels, due to defective autonomic control of the vasomotor mechanism. An objection to this is that it has not been proved that there is any active vasomotor control of cerebral vessels.

Cushing noted that epinephrin painted on the pial vessels blanches them, locally, just as does faradization. Sargent⁶ and Sherrington, together, observed that, when a faradic stimulus of strength sufficient to cause an epileptiform seizure was applied to the brain cortex of human beings and animals, the pia mater could be seen to blanch. One

can hardly call this satisfactory proof of vasomotor control of cerebral blood vessels. Epinephrin⁸ has the power to constrict vessels stripped entirely of nervous connection. In faradization, there are undoubted chemical changes which take place in the propagation of electrical stimuli, the results of which may act locally on the vessel walls just as does epinephrin, independently of any action of the alleged sympathetic-autonomic control. At any rate, Sargent makes the important deduction that vasoconstriction is the basic principle underlying the convulsions in the cases of traumatic epilepsy on which he operated.

All of these patients manifested epileptiform attacks soon after severe craniocerebral injuries. These injuries resulted in strong and rather extensive adhesions between the scalp, the meninges and the brain. The pathologic variations in the vasomotor control of the cerebral vessels in these cases cause rapid changes of vascular supply, which are sufficient in themselves, he thinks, to explain the recurrences of the convulsive phase of epilepsy. The severe manias, the deliria, the confusions, the attacks of petit mal of the Jacksonian type, all of which may occur without the characteristic loss of consciousness, etc. of the typical epileptic attack, cannot be explained in this way. Neither is the mental deterioration of advanced epilepsy satisfactorily explained. Therefore it is hazardous and even unnecessary to say that "it may be that the search (for the cause of idiopathic epilepsy) transferred from the barren field of neurohistopathologic investigations of the cortex to that of the autonomic nervous system, will prove more fruitful." Such a statement should have awaited absolute proof that the autonomic nervous system controls the cerebral circulation. This is especially true in view of the certain knowledge which we possess⁸ that the products of metabolism have a very definite action on peripheral blood vessels. Neither should one too hastily discard as of no value the work of Alzheimer¹⁰ and others on the neurohistopathology of the cerebral cortex in epilepsy.

It is important to review briefly the results of stimulation of the cortex of human and animal epileptics. The most important observations are those of Fedor Krause (1911), Sherrington,³ Sargent,⁶ and Gordon Holmes, on individuals suffering from Jacksonian fits, without gross visible lesions discoverable at operation. It was found that faradization of the epileptogenous zone alone elicited the fits, while stimulation of the rest of the cortex produced only the appropriate movements, even though the nature and duration of the current was the same. In Sherrington's monkey³ the epileptogenous areas were the tongue area of both sides, but the attacks were much more easily provoked from

10. Alzheimer: Ein Beitrag zur pathologischen Anatomie der Epilepsie, *Monatschr. f. Psychiat. u. Neurol.* 4:367, 1898.

the right side. A minimal faradic stimulus applied over the tongue area of the right cortex produced epileptoid seizures beginning in the tongue, which continued after the stimulus was withdrawn. These seizures, in response to faradization, never extended beyond the face and tongue. The animal's spontaneous attacks, however, were left-sided and, occasionally, generalized. It was impossible, in either the human or monkey epileptic, to provoke a Jacksonian attack of any magnitude. The limbs never took part in the seizures. This is explained on the theory that the depth of the narcosis used prevented the spread of the convulsions. This explanation however is not adequate, since something was lacking under the experimental conditions which was present when the subjects had their attacks spontaneously. This hitherto unknown influence accounts for the difference in the type of convulsive seizure. The probable nature of this factor will be discussed later.

In the experimental work of Sherrington, Krause, Horsley, Leyton, and others, one finds great agreement in the conclusions. First, there is a wide variation among animals in the ease with which the spread of the Jacksonian-like seizure is produced by faradization. Second, the excitability of the cortex is increased by repeated stimulation, so that a weaker current will evoke a spread. Third, the area from which a given movement may be obtained can be extended considerably by repeated stimulation. These facts were confirmed in the human being by Gordon Holmes and Sargent,⁶ who faradized the cortex in front and behind the Rolandic fissure in the case of a man suffering from Jacksonian epilepsy which was caused by craniocerebral injury. As a result of this experiment, these observers concluded that the movements caused by electric stimulation of the cortex are like the movements of the epileptic attack, in that they are possessed of the quality of purposive coordination (the fractional movements of Sherrington), but are not completely purposeful. A Jacksonian fit is a series of these fractional movements, devoid of mutual purpose, and "determined in point of sequence only by the relative anatomic position of cortical areas in which they are represented . . . so that the tongue may be bitten, due to the unnatural sequence of tongue protrusion and jaw closure."⁶ In this regard, the movements caused by electrical stimulation are exactly similar to those of the epileptic convulsive seizure. No purposeful movement, however simple, can be brought about by faradization of the cortex. This discussion eliminates the interpretation of the psychanalysts that the epileptic fit is a subconscious, symbolically purposeful act.

Sargent believes that the early fits which follow head injury are due to acute vascular disturbances: small hemorrhages, edema, injury to cortical tissue with inflammatory reactions, the effects of recurring sepsis, etc. The adhesion of the scars, binding the scalp to the brain

tissue, is the vital factor in those cases in which the attacks occur a long time after the injury. The initial disturbances of the vascular supply are caused by traumatic thrombosis and strangulation of vessels in the course of the severe scar formation in the cortex, and between the cortex and the scalp, or by adhesions between previously infected membranes and the cortex. These infections occur at the time of the infliction of the wound. After the acute phase, during which attacks are apt to recur frequently, there is a prolongation of the intervals and the explanation for the attacks must be sought in the vasoconstriction which is locally produced when the brain is pulled upon and distorted by the adherent scalp.

Normally every time the head moves the brain moves within the skull. This has been proved by frozen sections and by a kinesiometer. This movement is made possible by the space which exists between the dura and the arachnoid. When the brain is anchored to the scalp by adhesions, this movement is restricted, and there occurs at the point of anchorage a pull upon the brain which "mechanically or possibly by causing reflex vasoconstriction" produces a "momentary local anemia in the damaged brain and so initiates a fit."⁶

This reasoning, while it appeals rather strongly at first, leads to certain questions. Sargent reviewed over eighteen thousand cases of craniocerebral wounds with skull defect, which resulted from injuries sustained in the late war. Of these approximately only eight hundred (4.5 per cent.) developed epilepsy. Why? The answer is that "a given stimulus is not effective for all brains and it is necessary to assume in those patients who develop fits, a tendency to epilepsy," from what is vaguely called, "a relatively low degree of stability of the nervous tissue."⁶ A case is cited as an example of what Sargent means by his vague characterization. This case is also important from the standpoint of the effect of prolonged emotional situations and fatigue (psychogenic factor) in the production of epileptic seizures.

The patient was a man of 35, who had been wounded in the forehead at the age of 15. He was operated on for depressed fracture immediately, with a resultant large craniocerebral defect. He remained well for twenty years until he joined the British Motor Transport Service in France in 1915. After six months, he had his first general convulsion, and then two dozen between that time and the date of his admission to the hospital. The brain bulged through an irregular opening in the right frontal bone. At operation, in May, 1920, the scalp was dissected away from the dura and the latter was separated an inch or more from the bone around the edge of the opening. As in most of the cases of this sort operated on by Sargent, he placed a thin sheet of celluloid over the dura, between it and the inner surface of the skull. A thicker sheet of celluloid was used to close the opening. Ten weeks after the operation, the patient had one attack and has had none since—(Sargent's paper was read Oct. 13, 1921).

The operator's interpretation of this case is that "although for twenty years this patient had a cranial defect through which the brain was anchored to the skull, the local lesion had been insufficient to cause a fit until nervous stability had been lowered by the stress of warfare." It seems, however, that a more complete explanation would have to consider conceptions of Clark concerning the psychogenic origin of epilepsy, and those of Cannon and Crile based on their work on the conditions of painful emotions, fear, anger and fatigue. It may be that not only epinephrin but also other toxic substances are produced in the severe affective states mentioned and in fatigue, which give rise to the convulsive phenomena of epilepsy.

The proteoses which Cuneo¹¹ names as the convulsion-producing substances in epilepsy, are capable of causing epileptiform attacks when painted over the motor cortex. Cushing has shown that epinephrin may cause the same phenomenon. Lactic acid, a prominent abnormal constituent of the blood of individuals after excessive muscular exercise or under conditions of deficient oxygen supply,⁸ causes convulsions when injected intravenously.¹¹ Whatever the toxic agent may be, there certainly is some more tangible factor than that of the "stability" of the nervous system, which determines the presence of epilepsy in individuals who have suffered craniocerebral wounds. If this were not so, it would be hard to explain why only 4.5 per cent. of a large number of these cases have developed epilepsy.

Reed¹² undoubtedly had, in the light of Cuneo's work, a useful and helpful thought regarding the pathogenesis of the epileptic seizure. He was partly correct when he placed the causes for the origin of the epileptic attacks in the intestinal tract. According to Cuneo, however, the trouble is to be found in disordered carbohydrate metabolism in the small intestine and in the liver. Reed, incriminated the large gut, and with his co-workers insisted that all the pathology of epilepsy was contained therein. They therefore resected the large intestine in epileptics with much confidence and assurance. Much has been said concerning pathologic changes in epileptic brains, and neurohistopathologists have recently made important contributions to this subject. It seems certain, however, that the primary pathologic change is chiefly biochemical and metabolic, the changes in the brain structure being secondary. There is no lack of analogous situations in other central

11. Cuneo, Gerolamo: Biochemical and Biological Researches on the Pathogenesis of Epilepsy, *Riv. sper. di freniat.* **45**:408 (Jan. 31) 1922; Biochemical and Biological Researches on the Pathogenesis of Epilepsy, *ibid.* **40**:837 (Nov.) 1914.

12. Reed, A. L.: Diagnostic Methods and Pathologic Constants in Idiopathic Epilepsy, *J. A. M. A.* **66**:336 (Jan. 29) 1916; Constipation and Intestinal Infection in Epileptics, *ibid.* **67**:1157 (Oct. 14) 1916.

nervous system disorders. In at least one very well understood condition, Wilson's disease, there appears to be an intimate connection between the pathologic changes in the liver and those in the brain. There, too, a toxic substance of unknown nature seems to have originated in the liver, or at least to have left its most damaging effects there and in the basal ganglions, and to have produced less severe damage in other parts of the brain, including the cortex.

Cuneo began his work in the laboratory for experimental pharmacology in the University of Genoa, in 1915. Since that time, he has devoted himself continuously to this problem and only recently has begun to seek cooperation in applying his conceptions practically in a therapeutic way. He first demonstrated the presence of a toxic substance in the blood of epileptics who had recently had a convulsion. He identified this substance with the proteoses. It was not found in the blood of normal individuals nor of those ill with other conditions, and never in epileptics in the intervals between attacks. He showed that the urine of epileptics who have recently had a convulsion, contains a total organic acidity of 80 per cent instead of the normal 5 per cent. In some cases these ammoniacal nitrogenous salts were found to constitute even 100 per cent. of the total acidity in the urine. He demonstrated in epileptics a retention of total nitrogen, an increase in the nitrogen content, and continuous acidity of the feces.

In order to explain these phenomena, he investigated the chemistry of protein metabolism. In the normal individual, organic acids are oxidized and are capable of reduction to carbon dioxide and water. Neither they nor the proteoses are ever found in the normal blood. The problem became more important to solve when he found that the proteoses introduced into the blood of experimental animals produced convulsions, strictly comparable to those of epilepsy.

Proteoses exist only in the gastro-intestinal tract as a result of the process of digestion of proteins. What happens when they are changed during further protein metabolism, is not clear. Cuneo, at first, was tempted to assume that because of altered absorptive properties in the intestines, the proteoses entered the circulation unchanged. In testing out this theory of the proteosemia (albumosemia) of epilepsy, Cuneo administered a protein rich diet (meat almost exclusively), to one large group of epileptics, and, to another, a preponderatingly carbohydrate diet. If it was true that in epileptics, there existed a defect which made it possible for the intestine to absorb proteoses in an unaltered state, Cuneo argued that those patients on a meat diet should become decidedly worse, while those placed on the carbohydrate diet would be expected to improve. Exactly the opposite took place. The epileptics on a protein diet improved, the acidosis practically disappeared, the attacks became less severe and occurred at longer intervals; while

those on a carbohydrate diet had an increase (percental) in the acidosis, and the number and severity of convulsions were greater. It was plain, therefore, that the protein metabolism was not responsible. This view was further supported by the observation that peptones, another product of protein metabolism, were never found with the proteoses in the blood of epileptics. The next step was to clear up the question of the connection between the formation of proteoses and the intense acidosis, and the possible relation of both to the ingestion of carbohydrates. The author experimented with monosaccharids (glucose), disaccharids (cane sugar, lactose, maltose) and polysaccharids (starches). He found that while glucose, cane sugar and lactose, given with milk, do not produce acidosis, the starches administered as rice, paste, bread, chestnuts, potatoes, etc. produced an obvious increase in the elimination of organic acids in mild cases and an overpowering increase in severe cases. This, Cuneo explained by the fact that glucose is absorbed as such, without transformation; the disaccharids divide promptly by fixation of one molecule of water, into two molecules of sugar and are thus absorbed as glucose. The polysaccharids, however, being amorphous and having a higher molecular content, must be reduced, by the action of amylolytic ferments, to the simpler disaccharid, maltose, which, in turn, is divided by the action of maltase through hydration into two molecules of glucose before absorption can occur. The cause of the acidosis of epilepsy, therefore, must be sought in this first phase of the metabolism of starchy substances, that is, in the stage when the starch is transformed into maltose. The second stage, namely, the transformation of maltose into glucose, goes on in a perfectly normal way. It is in this first stage that the organic acids are formed.

Cuneo points out that the formation of organic acids in this stage of the fermentation of starches, frequently occurs not only in the process of digestion, but also, for instance, in the industry of brewing beer. Great care is taken to prevent the formation of these acids, because their presence causes turbidity, and a diminution of the alcohol content. The offending organic acids are acetic, butyric, lactic, tartaric, etc. Their production is favored by an insufficient amount of amylolytic ferment and may occur even in normal individuals, but the final disposition of these acids is different in the epileptic. In normal individuals, the acids are rendered innocuous, because they are changed into salts by the alkaline substances found along the paths of absorption. They are then oxidized into ammonium carbonate, which, when dehydrated, becomes urea if the salts are ammoniacal; or if they are sodium salts, the organic acids are changed into sodium carbonate. This is the substance which serves to maintain the alkalinity of the intestines.

These acids or acid organic salts which are transformable into carbon dioxide and water, are not normally eliminated as such, but are oxidized into urea and sodium carbonate. In the epileptic, however, this does not occur, for the organic salts are found unoxidized in the urine. Thus, this very important source of alkalization, does not develop or is deficient. As confirmation of this point, Cuneo points to experiments in which he administered equal doses of sodium acetate and tartrate to normals and epileptics who had constantly acid feces and urine from intestinal fermentation. He found that in the non-epileptics the acidity rapidly disappeared, while, in the epileptics, it increased and sodium acetate and tartrate were found in the urine. Clinically, the epileptic attacks became more frequent and severe. Normally these salts should disappear by being transformed into carbon dioxide and water. In fact, these patients were made so much worse, that the entire experiment had to be discontinued. It seems proved that in epileptics there is a deficiency in the antitoxic alkalization function, as a result of which organic acids transformable into carbon dioxide and water, and which normally are oxidized into urea or sodium carbonate, are not so changed. The organism is constantly in need of sodium carbonate; absence of it quickly causes death by paralysis of the respiratory and vasomotor centers. Animals treated intravenously with hydrochloric acid and other acids will die in this way. The intestine is always alkaline, in contradistinction to the stomach, which is always acid. The formation of sodium carbonate, therefore, is vitally important, not only because the organism cannot live unless the blood and tissues are alkaline, but also because intestinal digestion, which is essentially the transformation of starch into maltose, cannot continue in an acid medium.

If acids or organic salts are not transformed into sodium carbonate, they enter the circulation, change the blood chemistry, and cause convulsions. Intravenous injections of acetic and hydrochloric acids or of sodium formate, acetate, butyrate, propionate and valerianate, cause convulsive attacks. The citrates and alkaline tartrates and other organic salts, produce severe convulsions when painted on the motor cortex. These organic salts, therefore, which are capable of causing convulsions, are precisely the ones which, in the epileptic, do not become transformed into sodium carbonate. Physiologists have never been able to locate exactly the organs in which this alkali-forming antitoxic process occurs. Cuneo studied *in vitro* the action of the intestinal mucosa on these salts, and used the pancreas, the liver and the bile. His conclusions, based on work done, are that this function of the transformation of organic salts into sodium carbonate begins in the mucosa of the intestines and is completed in the liver, whence the sodium carbonate is returned to the gut with the bile. He further noted that when the

content of sodium tartrate or acetate in contact with liver tissue was abundant, oxidization took place regularly and sodium carbonate was formed; but when these salts were present in small quantities, the reaction became acid because of a lack of bases. Much carbon dioxide developed and proteoses were found in the liquid under these circumstances. If the liver tissue was kept in contact with a 5 per cent. solution of sodium tartrate, the tissue remained unaltered for several days, the tartrate was transformed into carbonate, and other substances were found which he was able to identify as nucleinic acid compounds; these later, under certain conditions, became split into proteoses. But if the liver tissue was kept immersed in a physiologic saline solution, it changed rapidly and no nucleinic acid compounds or proteoses were found. Therefore, while sodium chloride is not capable of maintaining the activity of these ferments, sodium tartrate by means of the oxidization it undergoes, may not only favor this activity, but can also give rise to the formation of sodium carbonate. Because of this double function, these organic salts may not enter the circulation there, to manifest their toxic activities. To prove this point further, he introduced intravenously in dogs, solutions of sodium tartrate and acetate, and caused not only typical epileptic convulsions, but, immediately after the convulsions, he was able to recover from the carotid artery a substance which proved to be a proteose. He used the same research methods to identify this substance as when he first discovered the proteoses in the blood of epileptics.¹¹

The problem is not clarified yet, however, because there is no explanation forthcoming from these experiments, as to the components of the blood which may, under certain conditions, liberate the substances which cause the convulsions, namely, the proteoses. Cuneo was able to produce convulsions in animals by the intravenous injections of proteose derived from nucleoproteins. Proteoses are not only developed in the intestines as a result of the metabolism of proteids, but may be obtained from that fundamental constituent of all cellular nuclei, nucleohistone. This substance is found in very large quantity in, and is easily obtained from leukocytes. It is very complex and contains an acid phosphate, or nucleinic acid, and a basic substance or histone which has all the properties of a proteose. Nucleohistone under normal conditions, is a stable salt and is therefore innocuous; but, under special circumstances, this nucleoproteid divides into nucleinic acid and proteose, and the latter then manifests its toxic properties. Cuneo obtained this substance from leukocytes. He worked on the theory that these substances are produced by a process of oxidization. The blood serum was hydrolyzed and oxidized by a solution of hypochlorite of sodium. In this way, two substances were obtained which have the same chemical and biologic properties as nucleinic acid and

the proteoses. In an alkaline solution, these substances, one an acid and the other a base, remain united as nucleohistone, a salt. But, when the solution is made acid by the addition of acetic acid, with the formation of sodium acetate, a substance is precipitated which has all the properties of nucleinic acid. The most important indication of these is the ability to color the blood serum indigo blue with the resin of guaiac. There remains in solution, however, a basic substance which has all the properties of the proteoses.

Cuneo studied the biologic effects of intravenous injection of these substances obtained from blood serum. He found that nucleinic acid is not toxic, but the basic substance (the proteose) causes typical convulsive epileptiform attacks in dogs. Animals dying in status epilepticus induced in this way, show the same pathologic changes in the viscera, brain, and their coverings, as do human beings who die from albumosemia (proteosemia).

He lists the necropsy findings in animals as follows: Hyperemia throughout all the viscera with many hemorrhagic infarcts; scattered pulmonary infarcts; infiltration of all tissues with bloody serum; vast edema; infiltration of the meninges, endocardium pleura and peritoneum; the presence of noncoagulable blood with the color of prune juice; distended gallbladder; and dilated heart. Tanzi and Lugaro¹³ list the necropsy findings in human beings who died in status epilepticus, in practically the same way. The acute tissue change are: edema of the brain; hemorrhages and venous hyperemia in both the gray and white matter, but especially in the pia; frequent hemorrhages in the pons and bulb; acute changes in the cellular elements of the brain (swelling, chromatolysis, proliferation and swelling of neuroglia cells, and the presence of amoeboid glia cells); hypertrophy of the heart; hypoplasia of the large vessels; persistent thymus, etc.

In this connection, it is well to recall the work of Russell and the quotations from Sargent, which point to the vascular factor as important in the pathogenesis of the epileptic attack. The necropsy findings in human beings who died in status epilepticus show very definitely how severely the cardiovascular system is damaged. The necropsy findings in Cuneo's dogs dead from experimental proteosemia (albumosemia) were exactly similar, and, further, Cuneo points to the facts that the blood which he extracted from epileptics in status epilepticus, or immediately after severe convulsive seizures, was also of prune color and incoagulable, and contained proteoses.

Cuneo, therefore, believes that albumosemia exists in epilepsy. This is caused by an insufficiency of the alkali-forming function in the liver and the small intestine, by means of which the acid organic salts, which are transformable into sodium carbonate, do not undergo this metabolic

13. Tanzi and Lugaro: *Mal. Ment.* 2:421, 1916.

change, but enter the circulation and cause a division, under certain conditions, of the nucleohistone element of the nucleoproteids into nucleinic acid and proteoses. These latter remain free and exhibit their convulsive action.

Based on this theory, Cuneo has recently made clinical therapeutic experiments. In order to aid the digestion of carbohydrates, he has attempted to administer alkali by mouth, but he readily admits the difficulties encountered. These are caused chiefly by the changes which these substances undergo in the acid content of the stomach. Remembering that the two substances obtained from blood serum are not split off in alkaline solution, but remain combined as an innocuous salt, whereas in an acid solution they decompose into the innocuous nucleinic acid and the toxic proteoses, he injected nucleinic acid in an alkaline solution subcutaneously, hoping thus to tie up the proteoses in the form of innocuous nucleohistone. Since 1917, when these therapeutic experiments were first begun, he has also ordered special diets restricted in the amount of carbohydrate (starches). This, combined with the alkali administration by mouth, has apparently given very encouraging results.

Before attempting to harmonize in a synthetic way the foregoing data, consideration must be given to the anatomic facts. These are best summarized by calling attention to the findings of Alzheimer¹⁰ in cases of idiopathic epilepsy. His conclusions translated literally are:

Macroscopically, a wrinkled, finely roughened condition of the surfaces of the convolutions, with more or less distinct sclerosis of the upper surfaces of the cortex. Microscopically, increase of the glia with a striking tendency to the preservation of the normal arrangement. There is also a considerable loss of nerve fibers and ganglion cells in the cortex. Many of the ganglion cells appear diminished in size, while others show certain changes, the specific significance of which must be further investigated. This cellular degeneration is spread over the entire cortex. There are reasons for believing that the nervous elements are primarily involved. The histologic changes indicate the presence of epileptic dementia.

Nissl, Bleuler and others (quoted by Tanzi and Lugaro) agree that these changes are more marked with the greater degrees of dementia. It must be kept in mind that all of the pathologic material was from old, demented epileptics. There has been of late a strong tendency to give to these findings no primary importance in the pathogenesis of dementia epileptica. It seems very probable that they are secondary and due to: the effects of the convulsive seizures; the prolonged toxemia which results from various attacks of status epilepticus; the effects of intracranial injury caused by the falls during attacks, etc. Many pathologists who have reported these changes, have seemed to stress the fact that they were most marked in the cornu ammonis. There is perhaps

nothing more significant in this observation than the clinically well-known fact that there is a type of hallucinatory epileptic seizure, often accompanied by convulsions, which has for its initial symptom a disturbance of the sense of smell or taste. A neoplastic or traumatic lesion in the rhinencephalon often gives rise to this type of seizure. As a general explanation for all the epilepsies, however, the localization of the gliosis and other pathologic changes in the cornu ammonis, has no more significance than the characteristic changes which occur in the same location in cases of senile dementia. No one has yet ventured to assert that all the mental phenomena of senile dementia are explained by the fact that the senile plaques and Alzheimer-Perusini neurofibrils¹⁴ are found preponderatingly in the cornu ammonis and in the tips of the temporal lobes. It is of interest to note that in pure senile dementia, uncomplicated by the excessive, specific vascular and ectodermal changes of arteriosclerosis, the convulsive or epileptiform attack is rarely seen. Certainly, convulsion is not a prominent feature in the senile psychotic picture.

If we are to consider epilepsy as a disease of the brain, it is strange that there are not more local signs of brain damage in the clinical picture. In looking over fifty-seven private cases of epilepsy, which have been carefully examined neurologically a number of times, only three of them showed any objective neurologic sign of disease or injury of the nervous system.¹⁵ Two of these were traumatic cases, and one was so-called idiopathic epilepsy. In the traumatic cases, there were definite signs of injury to the sensorimotor cortex. In the idiopathic case, there was a Babinski toe sign on the right, and a left external rectus palsy. These facts point away from actual brain change in any considerable proportion of cases of epilepsy.

A very brief mention of the bacteriologic theory of the pathogenesis of epilepsy is perhaps all that is merited. The idea that bacteria^{16, 17} cause epilepsy has never proved more than a fanciful notion. The work of those who advanced this as a fact not only has never been confirmed,

14. Bonfiglio, F.: *Anatomia patologica delle psicosi dell' eta senile*, Riv. sper. di freniat. **45**:219 (Jan. 31) 1922.

15. The question of aura in relation to "idiopathic epilepsy" has been held by many to stamp these cases as a definite entity distinct from other epilepsies. I was struck by the fact that many (twenty-four) of my private cases had no definite and constantly experienced aura at all, and all of the cases had some epileptic attacks without aura. The conception we advance explains the aura on the basis of stimulation, by toxic substances, of sensory areas shortly before the whole cortex is involved and unconsciousness supervenes.

16. Terhune, W. B.: A Bacillus Isolated from Epileptics, *J. A. M. A.* **67**: 1155 (Oct. 14) 1916.

17. Reed, C. A. L.: The Bacillus Epilepticus, *J. A. M. A.* **66**:1607 (May 20) 1916.

but Caro and Thom¹⁸ disproved the claims almost as soon as they were made. Finally, it is important to consider the question of heredity and hereditary factors in idiopathic epilepsy. There are those¹² who believe with Pierre Marie that "the cause of epilepsy exists always outside the patient and operates prior to his conception." This implies that prenatal encephalitis, trauma, infection and intoxication, defective cardiovascular development, etc. are always operative in the causation of epilepsy. On the other hand, some authors are less definite, and incline to the belief that, in some sort of Mendelian manner, epilepsy is directly inherited as a recessive trait of the nervous system. However that may be, it remains undisputed that numerous statistical studies have failed to show anything definitely tending to substantiate these ideas. Grouping epilepsy with all other mental disorders, the search for hereditary stigmata has been successful in from 66.8 per cent by Dejerine (quoted by Tanzi and Lugaro) to 87 per cent. by Kraepelin. When epilepsy in the ascendants is considered alone in these studies, and a search is made for similar heredity, the results are disappointing, and even exhaustive statistical studies show how negligible this factor must be. Burr¹⁹ has recently conducted such an investigation in a large series of cases with results which are barren for the notion that epilepsy is an hereditary trait in predestined individuals. Besides, no conception of the pathogenesis of epilepsy based on heredity alone, could possibly be made to explain all the epilepsies (traumatic Jacksonian attacks, general paralysis, arteriosclerosis, status thymicolymphaticus, brain tumor and cysts, alcoholism, etc.).

The sweeping statement of Pierre Marie, however it may fail to fit every case of epilepsy, does nevertheless explain a small group, especially the epilepsies of mentally defective and paralytic idiot children. His conception in these cases has undoubted validity. As with each of the theories of the pathogenesis of epilepsy above detailed, however, it leaves the subject as a whole, practically untouched. Not one of these conceptions, not even the vasoconstrictor vascular hypothesis of Sargent, or the biochemical explanation of Cuneo can be made to apply to all epilepsies.

Formulated briefly, our conception is that the research work of the future must be directed to the task of finding facts to substantiate and amplify the ideas of Cuneo. A defect in the alkalization function of the small intestine and liver in the process of carbohydrate metabolism, sets free in the blood toxic substances which, when they reach the

18. Caro, H., and Thom, D. A.: A Bacteriologic Study of the Blood of Seventy Epileptics with Especial Reference to the *Bacillus Epilepticus* of Reed, J. A. M. A. **67**:1088 (Oct. 7) 1916.

19. Burr, C. W.: Heredity in Epilepsy. Arch. Neurol. & Psychiat. **7**:721 (June) 1922.

brain, are capable of causing convulsions. In the idiopathic epilepsies, the proteoses or whatever the toxic substances may be, act on the larger cerebral vessels, as epinephrin or any acid does, and cause a sharp vasomotor disturbance with loss of consciousness. It will be recalled that proteoses like many other toxic substances, cause convulsions when painted on the motor cortex. With the large cerebral vessels constricted, the offending substance is imprisoned in the cortex and causes the generalized clonic convulsive phase of epilepsy. In the smaller attacks, the constricted area may be sharply circumscribed, as in urticaria or so-called angioneurotic edema. Thus we may explain the Jacksonian seizures. After a local vasoconstriction a transient hyperemia often follows, with transudation and the local liberation of toxic substances in the tissues. Lactic and other acids cause vasoconstriction in conditions of muscular exertion with diminished oxygen supply (asphyxia, violent exercise), or when injected intravenously.⁸ In the brain, convulsions may be reproduced experimentally by painting the cortex with proteoses or other toxic substances. In the case of craniocerebral injury with scar adhesion, brain abscess, circumscribed areas of encephalitis, brain tumor, cysts, general paralysis, cerebral arteriosclerosis, etc., the toxic substance may be produced locally as a result of the vascular disturbances. Cuneo has shown that the breakdown of leukocytes and all cellular tissue releases the nucleohistone which decomposes under certain circumstances (acid mediums experimentally) into nucleinic acid and proteoses. Any cell deprived even momentarily of its blood supply may become acid. The local liberation of proteose in the cortex in all these conditions would again give this convulsion-producing substance an opportunity for activity. Disturbed vasomotor control locally, as an autonomic system defect, is an added possibility which must await definite proof. However, the smooth muscle of blood vessels has inherently a capacity to react to various substances by manifesting vasoconstriction or vasodilation. This may be caused by excessive nitrogen or carbon dioxid in the blood, as in asphyxia. In the spinal animal (cord divided below the medulla to remove the medullary influence on the vasomotor functions in the extremities) extensive vasoconstriction with spasm of the skeletal muscles occur and cause a marked rise of blood pressure when lactic and other acids are injected intravenously. Lactic acid is only one of a number of acids capable of producing this phenomenon.⁸

Starling⁸ says in this connection that "in the absence of any direct histologic evidence of the existence of such centers (nerve centers in the walls of the vessels) it seems more rational to ascribe the tonus in peripheral blood vessels to the automatic activity of the muscular fibers themselves." The cerebral vessels in particular seem to be free from vasomotor control. This being so, the local liberation in an acid medium

(epileptic acidosis), or by the destruction of cellular tissue (leukocytes, ganglion and neuroglia cells, etc.), of nucleinic acid and proteoses will supply all of the conditions necessary for the production of the epileptic convulsion. The vasoconstriction might be caused by several toxic substances: proteose in so-called idiopathic epilepsy; epinephrin or some other substance in the so-called psychogenic (emotional factor) cases. In general paralysis and arteriosclerosis, the vascular disturbances and the local cellular destructive processes in the brain elements are ideally combined to produce generalized convulsive seizures. The periodicity of epileptic seizures in idiopathic cases would be explained by the occasional synchronous repetition of all the processes which produce the elements necessary to cause a seizure. The alkalization function may be sufficient to prevent the entrance of free organic acids or organic salts into the blood until the proper carbohydrate metabolic balance is upset, and causes an overwhelming acidosis and toxicosis, with the production of albumosemia (proteosemia). In cases where this condition obtains continuously for considerable periods, the status epilepticus supervenes or there is a fierce, sharp, mental epileptic equivalent or an even more prolonged and severe epileptic psychosis. No mental reaction is more severe, senseless, brutal or so apparently without reasonable basis, than the violent epileptic psychotic outbreak. The confusions¹⁴ (aura) and deliria preceding or following even the ordinary epileptic attacks, are very similar to the toxic deliria or the deliria of toxi-infective exhaustive states. One, therefore, can make this conception satisfactorily fit all of the above marshalled data.

Regardless of whether the vasoconstriction or other vasomotor disturbance is caused by the adherent scar in a craniocerebral wound, by an acidosis, or by local disturbance of a theoretical autonomic vascular control of cerebral vessels, it must be remembered that brain cells particularly cannot long survive a cutting off from their blood supply. Even when this is momentary, catabolic changes begin to occur. In the breakdown which results if the cells are bathed in an acid medium, the nucleohistone breaks up into nucleinic acid and proteoses. The proteose then causes the convulsion. This is the explanation of such experiments as those of Pike.⁹

Therefore, the factor of importance in epilepsy is the acidosis, regardless whether this depends on a viciously functioning carbohydrate metabolism which causes a general toxicosis, or upon the local production of the toxic substance from disintegrating cellular structures, secondary to vascular disturbances. The effect is the same, the only difference is the extent of the convulsive reaction.

Obituary

ROBERT THAXTER EDES, M.D.

1838-1923

Dr. Robert T. Edes died Jan. 12, 1923, at the home of his daughter in Springfield, Mass., at the age of 84. For many years he had not been closely identified with medicine because of advancing years and the infirmities dependent thereon, rather than through lack of abiding interest in his profession. Owing partly to increasing deafness, which rendered it quite impossible for him to attend meetings with satisfaction, or in fact to engage in the ordinary social relations of life, he was forced to retire largely from the interests which his active mind otherwise would have followed to the end.

Dr. Edes was born in Eastport, Me., Sept. 23, 1838. He was the son of a Unitarian clergyman and was descended from English stock, his ancestors having originally settled in Charlestown, Mass., toward the end of the seventeenth century. He was graduated from Harvard College in the class of 1858, and three years later took his degree in medicine at the Harvard Medical School. The Civil War had already begun at the time of his graduation and he forthwith entered the Navy, serving throughout the war and ultimately retiring with the rank of past assistant surgeon. During the four years of his association with the Navy he saw active service particularly in the engagements about New Orleans. Following this period, he undertook a course of study in Europe, and later settled in the neighborhood of Boston for the practice of his profession. He quickly took a prominent place among the physicians of that time and successively occupied the chairs of *Materia Medica* from 1875 to 1884, and of *Clinical Medicine* from 1884 to 1886, at the Harvard Medical School. He also held the post of visiting physician at the Boston City Hospital in a department, since abolished, of nervous and renal disease. During this period which was probably the happiest and most successful of his somewhat checkered career, he established a reputation for erudition and skill in diagnosis which was the envy of his contemporaries. While at the height of his success as a medical practitioner and consultant in Boston, he felt it necessary because of the illness of his wife to change his residence to Washington. This was in 1886, two years after his attainment of the professorship of *Clinical Medicine* at the Harvard Medical School. The conditions, however, were such that after a stay of about five years in Washington he returned to Boston and forthwith became

resident physician of the Adams Nervine Asylum in Jamaica Plain, a suburb of Boston, an institution established for the care of nervous conditions and, essentially, what we should now call "border-line" cases.

Dr. Edes, although versed in general medicine, and by no means a specialist in any narrow sense, nevertheless always took a very vital interest in disorders of the nervous system, both organic and functional, and became known as an authority for that time in this type of disorder. It was natural, therefore, that he should identify himself with organizations devoted to the special study of the nervous system. Although not one of the founders of the American Neurological Association, he became one of its earlier and most devoted members, resigning later when he found it impossible to take further active part in its deliberations. The Adams Nervine Asylum, a comparatively small hospital, did not offer full scope for the capacities of his really fertile and original mind. He remained at its head, however, for a number of years, practiced to a certain extent privately, later was a visiting physician to a sanatorium in Nashua, N. H., and in 1903, moved to Reading, in the neighborhood of Boston, where he attempted to establish a small private sanitarium, particularly for nervous disorders. He was, however, through his deafness and through the progress of the years, losing somewhat his capacity to meet the exigencies of modern medical practice, so that after a period of waning success he retired completely from medical practice, and after a long period of semi-invalidism, which was the harder for him on account of his clear mental vision, he died at the ripe age of 84.

His life was rich in attainment; he had his share of the honors which his profession could bestow, but he was on the other hand saddened by various misfortunes of a personal sort. Among these stands out prominently the loss of his only son, Richard Edward Edes, a young man of great promise, who early in his career, at the age of 32, died after a lingering illness from tuberculosis. This son, educated at the Johns Hopkins University, the Harvard Medical School and Massachusetts General Hospital, was a man of exceptional attainment and promise. His death, coming at a time when his father had reached and perhaps somewhat passed the zenith of his career, was a blow from which he never recovered, which darkened the declining years of his life, but which he bore with characteristic equanimity and fortitude.

Dr. Edes had a large society membership, among the more important of which were the Association of American Physicians, the American Neurological Association, the Philosophical Society of Washington, the American Academy of Arts and Sciences and the American Medical Association. He was also a companion of the

Military Order of the Loyal Legion. He taught not only at the Harvard Medical School, but also, at different times, at Dartmouth on the subjects of *Materia Medica* and *Therapeutics*; while in Washington, he was a lecturer on diseases of the nervous system in Columbian University and at Georgetown University on renal disease. For many years an active member of the Massachusetts Medical Society, he was placed on the retired list in 1912. He was a ready and trenchant writer, and his papers and articles all bear the stamp of originality and clear thinking. Among his earliest ventures in the literary field was a paper written in 1868 on "The Part Taken by Nature and Time in the Cure of Disease," for which he was awarded a prize by the Massachusetts Medical Society. In this paper was foreshadowed his unwillingness to accept traditional views and generally accepted opinions, unless based upon most substantial foundations. He was always in the forefront of progress and often distinctly ahead of the thought of his time. For example, in this "dissertation," as it is called, the following passages occur: "The more philosophical spirit of modern inquiry, which leads us to look at things as they are, and not as we would have them to be, is causing scientific men to diminish their estimate of the physician's power over the natural processes of disease . . ." and further, "The medical profession of the present day, though possessing far greater knowledge of physiological and pathological science than ever before, and far greater resources in the arts of medicine and surgery, must yet be content to acknowledge the somewhat narrow limits of its curative powers, and while really doing much more, can honestly claim as its own work much less than earlier and less instructed generations thought they had a right to do." This was written fifty years ago and shows the healthy scepticism, since familiar enough, which, in the minds of the pioneers, was beginning to rouse the medical profession from its complacency. Dr. Edes' interest soon turned to the nervous system and there appeared from his pen, in a relatively short time, upwards of a dozen articles on brain tumors and about as many more on various other disorders of the nervous system. These, however, were interspersed with papers on anemia, cancer and renal disease, the latter, also, a subject in which he was vitally interested. His interest in physical therapeutics found expression in a handbook published in 1883 and later, in 1887, in a work on *Therapeutics and Materia Medica*. In 1885 he gave the Shattuck lecture before the Massachusetts Medical Society on "The New England Invalid." This, like his earlier paper, to which allusion has been made, foreshadowed with extraordinary perspicacity the modern trend in the treatment of the psychoneuroses. "The psychic element in invalidism is to be controlled by psychic means"; "Nervous invalidism is a psychosis, and the study of it is a branch of morbid

psychology." This lecture is a notable contribution to our knowledge of this complex field, written in a style of scientific exactitude and whimsical humor in rare combination. Up to the time of his death, although handicapped in other respects and perforce a recluse, his mind remained clear and he continued to write for various periodicals almost to the end.

Although buffeted by Fate more than most men and destined to drag out many years of his prolonged life in comparative inactivity, Dr. Edes stands out as a man distinguished among his fellows. Scientific ardor, a delightful sense of humor, geniality and clear mental vision were so happily united in him, that while recognizing his great accomplishment, one wonders what more he might have achieved had the circumstances of his life been more favorable. After his return from Washington in the early nineties he was unable to reestablish himself in a position commensurate with his sterling abilities. The world moved on and, in a material way, he was not able to keep pace with it, in spite of his intellectual acumen and unfailing interest in its progress. Few of his contemporaries are living, but, to the younger generation who knew him, his memory will be cherished as one of the outstanding figures of his time.

E. W. T.

News and Comment

AMERICAN NEUROLOGICAL ASSOCIATION

The forty-ninth annual meeting of the American Neurological Association will be held in Boston, May 31, June 1 and 2, 1923.

AMERICAN PSYCHIATRIC ASSOCIATION

The annual meeting of the American Psychiatric Association will be held June 19 to 22, 1923, inclusive, at Hotel Statler, Detroit, Mich.

Abstracts from Current Literature

SPIROCHETES IN THE CENTRAL NERVOUS SYSTEM IN GENERAL PARALYSIS. F. JAHNEL, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **73**:310, 1921.

Jahnel begins with a summary of the findings of Valente, a Portuguese investigator, who made brain punctures on cases of general paralysis after the method of Forster and Tomaszewski. He found spirochetes in 70 per cent. of the cases. In several he found spirochetes only after a second puncture, thereby proving that failure to demonstrate spirochetes does not prove their absence. There was no relationship between the finding of spirochetes and the stage of the infection, the duration of the paralysis, or the clinical form. In three cases he found spirochetes although the spinal fluid gave a negative Wassermann reaction; in four cases the Wassermann test was doubtful and twenty-one times it was positive. There were no morphologic or staining peculiarities of the spirochetes. The histologic examination in these cases gave a typical picture of general paralysis. In one case the spirochetes were extraordinarily numerous; they were never found in the pia, were numerous in the molecular layer and present even in the membrana limitans. The spirochetes were found in largest numbers in the second and third cortical layers. They were only rarely found in the white substance. In the neighborhood of the spirochetes, Valente found marked cell loss and a considerable new growth of vessels. Nevertheless, in some places with most marked tissue change, there were relatively few spirochetes. Valente believed that the organisms were not equally distributed throughout, but that in different places there were groups of spirochetes. Contrary to the findings of Noguchi, Valente demonstrated spirochetes in the neighborhood of the vessels and lymph sheaths, at times even in the lymph sheaths of the large vessels which enter the brain substance from the pia. Valente never found spirochetes within the plasma cells, although he found them between plasma cells. Groups of spirochetes were also present around the ganglion cells. He attempted to infect rabbits with puncture material from living general paralytics as well as with blood and spinal fluid; but in spite of many attempts in 103 animals he had only negative results. (In the clinic of the reviewer negative results were also obtained.) Valente concluded, as had Forster and Tomaszewski, that the virulence of spirochetes from general paralytics was reduced for the rabbit. He considered the paralytic process as the product of local spirochetal effects in the central nervous system. He inclined to the opinion that general paralysis develops from the early syphilitic meningitis, and that the spirochetes, in some manner still unknown, break through the limiting glial membrane.

Jahnel says that every variety of spirochetal finding in the brain of syphilitics is possible. Sometimes they are present in large numbers; sometimes only in a few places; sometimes in many areas throughout the brain. Cases in which one can find many spirochetes in the brain are relatively rare. Still they can be found in a great many cases, probably at least 50 per cent. In brain punctures, Forster and Tomaszewski found spirochetes in 47 per cent. and Valente in 70 per cent. In those cases in which they were not found one

must remember that the whole brain was not searched. In those cases in which death follows a paralytic attack they are usually found. He states that Ehrlich advanced the theory that the spirochetes in general paralysis have an increased resistance due to the fact that, after invasion, antibodies are formed which kill all but the resistant spirochetes. There thus arises a strain with increased resistance, which will again call forth more antibody formation and again kill all but the resistant ones, and so on ad infinitum. However, there is no proof of the validity of this theory. Jahnel then presents the theory of a neurotropic strain and calls attention to the work of Levaditi and Marie. Jahnel is not quite ready to accept their views, and among other things calls attention to the fact that there is a form of rabbit spirochetosis which may at times occur and thus spoil the value of the experiment.

He calls attention to the great rarity with which spirochetes have been found in the internal organs of adult syphilitics as contrasted with congenital syphilitics. The work of Warthin in finding spirochetes in a large number of cases is novel. Jahnel himself says he has searched through the internal organs of a number of general paralytics without finding spirochetes. Once he found spirochetes in the lung, and thinks they may have made their way there from the brain. He also found spirochetes in the aorta and presents a photomicrograph of them.

He agrees with Noguchi that in general paralysis spirochetes are found chiefly in the cortex of the brain. He has occasionally, though rarely, found them in the white substance. In the cortex the spirochetes are generally located in those areas in which the general paralytic process is recognized to be most frequent. They are found most frequently in the region of the frontal pole. However, they are also to be seen in the central convolutions, in the temporal, parietal, and, least frequently, in the occipital region. He has also found them in the optic thalamus, in the cerebellum, and in the midbrain, which is of interest in relation to the pupillary abnormalities which are so common in general paralysis. He states that there is a very great difference in the number of spirochetes to be found in different brains. He feels that there are some brains in which long search will not reveal spirochetes. There are others in which they are present in very great numbers. This does not agree with the uniformity of the findings in the cerebrospinal fluid, and leads Jahnel to believe that there may be a different form of spirochete.

Jahnel says that he has found spirochetes in places where they had not previously been found; in the meninges of the cerebellum, in the midbrain, in the walls of the basilar vessels and in several cerebellar veins. Generally, however, the spirochetes are found only in a few places. The spirochetes have only been found in the meninges occasionally; frequently in large numbers in the cortex. However, whenever Jahnel found spirochetes in the cerebellum he also found them in the meninges of the cerebellum. The question as to whether the cases in which spirochetes were found in the meninges might not be cases of cerebral syphilis rather than general paralysis is answered by Jahnel in the negative as the result of his microscopic examinations. It is to be remembered that in cases of true brain syphilis the spirochetes are present in very great numbers in the meninges as contrasted to a few in those cases of general paralysis in which they have been found at all. The finding of spirochetes in the meninges in cases of general paralysis makes a bridge between the cases of cerebral syphilis and general paralysis, and perhaps helps us in the question of preparalytic conditions and in the

therapy of general paralysis by showing that the battle must be not only against the spirochetes in the brain substance but also against those in the brain membranes.

As regards tabes, the finding of spirochetes is much more infrequent than in cases of general paralysis. Noguchi in his original study of twelve cases found only a few spirochetes in the posterior column of one case. Shortly after, Versé reported two cases of tabes in which spirochetes had been found: in one case they were in a posterior root; in the other case in a spinal ganglion; but he was not entirely sure that the objects seen were really spirochetes. Then Richter in a profound study of the histogenesis of tabes stated that the real point of attack was the nerve of Nageotte. He found spirochetes in the granulation tissue of the posterior root. Jahnel says that in many searches in the past he was unable to find spirochetes in the spinal cord, either in tabes or general paralysis, but more recently, when he made sections with the dura intact, he found spirochetes in the arachnoid tissue in different places, at times in groups, at times singly. He also found them in the membranes of the posterior root. He thinks that in removing the dura in the past, a good deal of the arachnoid tissue was torn away, and this accounts for having missed them. In considering the relationship between the finding of spirochetes in tabes and the pathogenesis of the disease as regards localization, it must be remembered that the anatomic preparations show the end-result of the process that has lasted for years, whereas the spirochete finding represents a condition that obtained at the moment of the death of the patient.

As to the spatial arrangement of spirochetes in the brain, Valente believes that they are more likely to be found at the base of the convolutions than at the top. They occur at times in small colonies in the center of which is a brown colorization, the cause of which is not clear. Another interesting condition is the presence of spirochetes in relation to the blood vessels. Jahnel thinks that this latter is more in the nature of colony formation. The finding of spirochetes in the vessel lumen, Jahnel considers to be the result of postmortem change; they have never been seen in the blood channels during life. The question of spirochetes in the spinal fluid is also in doubt. It is possible that when found as the result of animal inoculation they may have been forced into the fluid, and were not really growing there. In the brain cortex the spirochetes, in the majority of instances, are in the nerve cell layers and only rarely do they occur in the molecular layers. A relationship to certain tissue elements has not been proved. Occasionally, one finds ganglion cells surrounded by a number of spirochetes. The presence of spirochetes within the ganglion cell has been but rarely reported and is not certain. Jahnel feels that this is a matter of subjective interpretation; the same is true for the relation to glia cells.

This article is very well illustrated with photomicrographs which demonstrate the presence of spirochetes in the various portions of the brain. This work on spirochetosis in the brain of general paralytics is opening new channels for the study of the etiology and pathogenesis of general paralysis. The same is true for tabes. As a result of the improvement in staining technic for spirochetes which has been devised by Jahnel, great progress in this study has been made in the last two or three years, especially by Jahnel and his students Hermel and Jacob. In this country Dunlap has contributed a most interesting and valuable review in an article which was published in the ARCHIVES, December, 1922, page 589.

SOLOMON, Boston.

CLINICAL CONSIDERATIONS ON PARKINSON'S DISEASE FOLLOWING ENCEPHALITIS. G. CATOLA, Riv. d. Patol. nerv. e ment. 27:62, 1922.

The author believes that the Parkinsonian syndromes of epidemic encephalitis belong fundamentally with genuine Parkinson's disease. This conclusion is of great clinical importance as it implies the existence of an intimate relation of cause and effect between an infection, encephalitis, and paralysis agitans, and thus throws a bright light on the hitherto obscure and controversial problem of the etiology and pathology of this disease. Until now the most frequent causes of Parkinson's disease were stated to be trauma, acute and prolonged emotions, and cerebral arteriosclerosis; only a few authors have sought to connect this condition with various infections or intoxications. Of these the most frequently mentioned were plumbism, acute articular rheumatism, scarlet fever, typhoid and dysentery. Dana, in 1898, was the most insistent upholder of the idea that infections and intoxications were the cause of the condition regardless of whether they had existed a longer or a shorter time prior to the onset. The author very strongly disagrees with the idea that even grave trauma or emotion, no matter how violent or prolonged, have any etiologic or pathogenic influence on the production of the Parkinsonian syndrome.

It is only necessary to call upon the evidence accruing from the late war, with the immense numbers of severe organic and functional nervous system injuries, and to note that there has not been as a result an exceptional or even noteworthy increase in the number of Parkinsonian cases. The author believes that any increase of these cases caused by intense prolonged emotional stress or actual injury to the nervous system following the war would have been commented upon. The author feels that the histories cited by those who have reported Parkinsonian cases following trauma or emotional stress, do not stand critical analysis well. Besides, he fails to understand how a physical or psychic trauma can, of itself, induce anatomic lesions which have a chronic and progressive evolution which presupposes the continuous action of an agent over a long period. The notion that Parkinson's disease is caused by precocious, senile or arteriosclerotic changes in the nervous system, is not borne out by the presence of this condition in young children and youthful individuals.

The author refers to the cases of Ramsey Hunt, who separated a definite group of juvenile cases from the presenile and senile types, and placed them on a definite autonomic and pathologic basis. The author does not believe that Parkinson's disease has a different etiology from that syndrome following any infection which involves the corpus striatum and the locus niger. Any disease process operating in the area of the brain where modern research has placed the localization of the anatomical changes in Parkinson's disease, will give origin to a similar picture. In other words, it is necessary to determine whether Parkinson's disease is a syndrome depending on the presence in the same area of many diverse processes or whether it is a nosological entity always due to one cause. Eliminating those very rare cases caused by gross lesions such as tumors, softenings, cysts, etc., it is possible to sustain a theory that this condition is universally of an infectious origin.

The anatomic and pathologic investigations of Hunt, Tretiakoff, Souques, and others, have revealed that there are always demonstrable histologic lesions to be found in the striomesencephalic region. This part of the brain may be said to have a special affinity for many varieties of toxic-infections. However, this hypothesis does not appeal to the author because of the relative rarity of Parkinson's disease, as compared with the frequency of the infections and intoxications. Others advance the theory that the special vulnerability of this

region is congenital or acquired thus making for a locus of less resistance to varied types of infections. This supposition would have great weight if there existed a more obviously direct relation of cause and effect between the development of Parkinson's disease and those infections which it has been held might be the cause of the condition. In looking over the histories of these cases, analysis fails to show an intimate and immediate connection. The author was surprised to find that the presumed causal infection had occurred at a time so remote that it was impossible to consider that any etiologic connection existed. He further observes that if the peculiar predilection as to the site of the disease were conditioned by exaggerated vulnerability due to pre-existing causes, it would be impossible to explain the percental difference in the occurrence of Parkinson's disease following infections in general and encephalitis in particular. The statistics of Barré and Reys show twenty Parkinsonian cases out of a total of one hundred and one cases of encephalitis.

The author stresses the point that it is exceptional to find an obvious connection between Parkinson's and any other infectious disease. He believes that it is logical to ascribe an elective localization to the virus of encephalitis and that, as a result, the pre-eminent infective etiologic cause of Parkinson's disease is always similar. The frequency of Parkinson's syndrome in epidemic encephalitis is compared to the epidemics of poliomyelitis, while the sporadic case of Parkinson's is comparable to the occasionally encountered case of Heine-Medin's disease in endemic form.

The author calls attention to the experience, which he shares with many others, that many Parkinsonian cases have followed *formes frustes* of encephalitis; some of these cases have been afebrile and ambulatory and the diagnosis was first made when the Parkinsonian features were noted. He feels that there is a strong connection between these attenuated types of encephalitis and the ordinary Parkinsonian picture. In fact, many months often intervene between the abortive attack of encephalitis and the onset of the Parkinsonian picture. The author also stresses the findings of Barré in 1910. This author found that many classical types of Parkinson's disease have shown weaknesses of the ocular muscles and diplopia. The author concludes that there does not exist any difference between the classical Parkinsonian syndrome and that following encephalitis and that the same infection may always constitute the direct cause of this picture. The isolated so-called classical types are caused by sporadic *formes frustes* of encephalitis epidemica.

This article was part of a symposium on encephalitis and was read at the annual congress of the Italian Neurological Society which was held at Florence, Oct. 19, 1921. It was also read at the reunion of the Neurological Society of Paris, June 1, 1921. At the Paris meeting the author's opinions were endorsed by Souques and other Frenchmen. He stresses the progressive, chronic, incurable nature of the postencephalitic Parkinsonian cases. OSNATO, New York.

REVIEW OF THE CLINICAL SEQUELAE OF THE RECENT EPIDEMIC OF LETHARGIC ENCEPHALITIS. F. NAVILLE, Schweizer Arch. f. Neurol. u. Psychiat. **11**:34, 1922.

The most frequent and important sequelae of encephalitis are the development of the Parkinson syndrome, pains, myoclonias, disturbances of sleep, and mental enfeeblement. Naville, in reviewing the history of the disease, states that in 1915, 1916, and 1917 Cruchet and Economo recognized an epidemic of encephalitis, which the former called subacute encephalomyelitis and the latter, encephalitis lethargica. In 1918 a form with predominance of medullary

symptoms appeared in Australia, also the myoclonic and Parkinson types. The epidemic subsided in the summer of 1920 only to reappear in the first months of 1921.

The incidence in Switzerland was 0.3 per ten thousand. It did not affect the urban population predominantly as in highly contagious epidemics. The greatest incidence was in people between the ages of fifteen and fifty years, although infants and people of eighty years of age were stricken. The mortality was 20 per cent.

Preceding epidemics had been reported by Hildestein in 1695 as sleeping sickness; in Tübingen, in 1713; as Dubini's chorea in 1846; by Gayet in 1875; and as nona in 1890. Little more has been learned concerning recurrences, chronicity, delayed exitus, and subsequent aggravation of symptoms than was known at that time.

In reviewing the symptomatology, Naville discusses diurnal somnolence, amnesias, sexual frigidity, apathy, syncope, vertigo, Parkinson states, insomnia, pains, chronic headache, visual disturbances, mental deterioration, general feebleness, fatigue, salivation, decrease or increase of appetite, obesity, modification of the genitals, and ocular disturbances. Grossmann, who followed a hundred cases of encephalitis from six to seventeen months after the acute attack, found a persistence of psychic disorders, as neurasthenia and hysteria, in 55 per cent.; involuntary muscular movements and tremors in 58 per cent.; disturbance of muscle tonicity in 18 per cent.; residual cranial nerve palsies in 64 per cent.; pupillary abnormalities in 30 per cent.

The Parkinson syndrome may develop after any type of onset. As a rule it begins within the first two months, but it may appear very insidiously many months after recovery seems to have been complete. The author reviews the various explanations advanced for the development of this syndrome, including emotional disturbances, muscular lesions, endocrine disorders, lesions of the locus niger, degeneration of the striothalamic fibers and of the ansa lenticularis, lesions in the globus pallidus and the efferent fibers in the body of Luys. Recent investigations seem to point to constant involvement of the locus niger, both in the classical Parkinson's disease and in the post-encephalitis Parkinson syndrome. Numerous theories regarding the physiopathology of these muscular disorders have been erected and are reviewed by the author. There seems to be no agreement.

In discussing the pains that sometimes accompany this illness Naville cites various views regarding their probable origin. In some cases pains appear to be radicular, in some sympathetic, in some meningeal, and in others medullary. They may appear in any part of the body, are often mobile, and may or may not be associated with muscular phenomena.

Acute chorea and generalized locomotor excitation and delirium have been common at the onset. Choreiform, clonic, rhythmic, fascicular, and ameboid movements are among some of the later manifestations of involuntary muscle movements. These movements may be continuous or may recur at more or less regular intervals, such as the *crises quotidiennes* of Harvier. On the whole, myoclonic movements present in the same individual are synchronous. Marie has called particular attention to simultaneous flexion or extension of a leg with coincident adduction of the arm and elevation of the shoulder on the same side under the euphemism *chorée saluante rythmique*. The maximum duration of these movements is not known. They seem to diminish in intensity, in amplitude, in frequency, and then disappear during sleep. They may resemble functional disturbances. On the whole their prognosis is more favorable than

that of the Parkinson condition. It seems that the former are irritative phenomena while the latter is based on a definite atrophic lesion. Sometimes a progressive cachexia with mental disturbance may supervene.

An interesting type of encephalomyelitis, in which myoclonic phenomena may appear, has been described in dogs. There are found diffuse vascular lesions in the cortex, brain, bulb, cord, and meninges. Chauveau has shown that if different sections of the cord of such an animal are made, each segment will continue to produce its own set of movements independently. This segmental explanation, however, will not cover all cases, particularly those in which there may be synchronous movements of one side of the face and of the opposite arm and leg. Some authors insist that all of these disturbances arise in the basal ganglions. Thomas has described a very instructive case in which these movements were synchronous with the cardiac pulsation. The fact that patients having the Parkinson syndrome sometimes complain of a subjective tremor which can not be seen objectively, has led to the hypothesis that these disturbances may have their origin on the sensory, rather than on the motor side.

The mental sequelae are very interesting and a paper on the subject by Naville has been published in *L'Encéphale*, June and July, 1922. In spite of the frequency of initial delirium, true psychoses which are prolonged do not often develop. Naville thinks there is something rather characteristic both in the initial delirium, often present without pyrexia, and in the later psychoses. In these he recognizes a characteristic lethargy, or psychomotor torpor. This raises the interesting question as to what component of this psychomotor torpor is physiologic and what psychologic. The question is a large one and has a bearing not only on this, but on other psychiatric problems as well, such as the influence of tics, choreas, professional cramps, psychomotor obsessions, psychasthenias, etc. He proposes the name *bradyphrénie* for this characteristic post-encephalitic mental state, so often present without Parkinsonian rigidity.

Delayed deaths are occasionally due to progressive cachexia in the Parkinson and in the myoclonic types, bulbar accidents, intercurrent diseases, and finally recurrences of the disease itself.

WOLTMAN, Rochester, Minn.

GENERAL PROTEIN THERAPY AND ACTIVE IMMUNIZATION OF
GENERAL PARALYSIS. W. JACOBI, *Ztschr. f. d. ges. Neurol. u. Psychiat.*
73:575, 1921.

Jacobi outlines an attempt to treat general paresis by the process of developing an active immunity toward the spirochete. He develops his theory as follows: at the time when specific chemotherapy for the so-called metasymphilitic conditions was forsaken (prior to the arsphenamin era) one recognized the occurrence of spontaneous remissions in general paralysis which, although occasional, were still a definite indication that a protective reaction (*Abwehrreaktion*) occurred which apparently brought the disease process to a standstill for the time being. The work of Gennerich has caused interest again to turn to immunity processes in the metasymphilitic diseases. According to Gennerich the development of these conditions has the closest relationship with the healing of the infection in the cerebrospinal fluid. The more generalized throughout the body the infection with the organism, the greater the production of immune bodies and hence the greater the probability of resisting the infection. The severe skin lesions in the beginning of the disease in many syphilitics are an expression of the increased resistance of the body in the sense of an allergic reaction, which apparently has a great relation to the later development of metasymphilis. Those cases which had marked skin lesions

very rarely develop tabes or general paralysis. If the protective reaction (*Abwehrreaktion*) is weak, some strains of spirochetes will remain behind in those places where the protective mechanism is especially poorly developed. According to Gennerich's view, this is especially true of the meninges. He believes that the course of the fluid infection is inversely proportional to the extent of generalization of the process, and that here lies the danger of anti-syphilitic treatment, especially with arsphenamin. When most, but not all, of the organisms are killed by arsphenamin, and the natural immunity is thereby reduced, there is danger of greater growth of the organisms unhindered by specific antibodies. In the majority of cases there is invasion of the central nervous system during the primary and secondary stages. Thus they are prepared for later manifestations.

Jacobi calls attention to the skin reactions which are produced in syphilitic persons by injection of syphilitic organ extracts. This "allergic serum reaction" occurs in gummatous lesions and in congenital syphilis but not in the metasyphilitic conditions. The appearance of this reaction tends to show that the infected person has a strong protective reaction. The failure of the skin reaction in the case of general paralysis leads Jacobi to deduce that the protective mechanism is lessened in this condition. He therefore assumes that, in these metasyphilitic conditions, which are after all active syphilis, there is reason to activate the immune responses. The fact that remissions occur indicates to him that it is possible for such immunity reactions to occur spontaneously. In regard to this reaction he uses the method for producing active immunity, namely injection of syphilitic material. For this purpose he used extracts of syphilitic fetal liver. Similar vaccine treatment has been tried in other stages of syphilis with varying result; some investigators believe that good results were obtained; others that the method was worthless. Jacobi made injections of extracts of syphilitic fetal liver in increasing doses in three patients. The first patient received 59 c.c. of the extract in forty-three injections. The second patient 55 c.c. in thirty-seven injections and the third patient 50 c.c. in thirty-eight injections. Febrile reactions and chills followed the injections. The first patient treated, aged 48 years, had an expansive type of general paralysis. There was no effect upon the serology, the psychosis became more marked, and he died about six months after the commencement of the treatment. The second patient, 49 years of age, had also an expansive type of general paralysis. The Nonne-Appelt reaction became negative, the cell count was reduced, and the spinal fluid Wassermann reaction became weaker. The patient improved and was able to go home. The third patient, aged 37 years, presented a simple dementing type of general paralysis. In this case, the Nonne-Appelt reaction became weaker, the cell count decreased and the man also developed a remission.

In summarizing the results of his half-year of experience in the treatment of these three cases, with remissions in two, Jacobi questions the reason for this result. If it is to be assumed that the remissions were the result of the treatment there still remains the question whether it was a specific or non-specific effect of the treatment. Recalling the results which have been claimed for non-specific protein therapy and infectious fevers as reported by Wagner-Jauregg, Döllken, Donath, Plaut, Steiner and others, Jacobi admits that his results may have been caused by the febrile reaction rather than as a specific reaction to the syphilitic extract. However, he feels that it is theoretically possible that the results may be due to the combination of a specific immunity with the effects of fever and leukocytosis.

SOLOMON, Boston.

A CASE OF CONGENITAL MEGACOLON (HIRSCHSPRUNG'S DISEASE) ASSOCIATED WITH MENTAL DISORDER AND TERMINAL CEREBELLAR HEMORRHAGE. N. D. C. LEWIS, *J. Nerv. & Ment. Dis.* **56**:193 (Sept.) 1922.

The patient was of Scandinavian race. The father was an alcoholic, and a brother and sister died of tuberculosis. At 15 the patient began to drink and continued the excessive use of alcohol for several years. At 23 he had typhoid fever and a short time later, while intoxicated, a homosexual experience. At the age of 27 he was imprisoned on a charge of train robbery, and after six or eight years was transferred to the government hospital because of insanity. The striking mental symptoms noted there were: auditory, visual, and gustatory hallucinations; delusions of persecution, and emotional indifference. At times he assumed peculiar attitudes and was then negativistic. The principal physical manifestations were epileptiform seizures and recurrent attacks of acute abdominal distention with visible peristalsis. During one of these latter attacks the patient died with manifestations of shock.

At necropsy the colon was found to be enormously dilated. Macroscopically, the liver and spleen showed interstitial changes. Acute and productive changes were noted in the kidneys. The right testicle was not found. There was a chronic interstitial myocarditis with hypertrophy of the left auricular appendage, some sclerosis of the aortic valve and of the arch of the aorta and terminal pulmonary congestion. The brain weighed 1,470 grains. The dura was slightly adherent to the pia; congestion and arteriosclerosis were present. There was a hemorrhage in the right lobe of the cerebellum which had destroyed the central cerebellar substance. Microscopically, the pituitary showed sclerosis with compensatory hypertrophy; the thyroid showed diminution of colloid and acini; the adrenals exhibited deficiency in the development of the cortex and hypoplasia of the medulla. The principal changes in the cerebral cortex were arteriosclerotic processes. The hypertrophied wall of the intestine showed an enormous increase of the inner and outer muscular bundles with fibrous and arteriosclerotic changes in the submucosa.

In his analysis of the case the author offers explanations for the patient's behavior which are based on the pathologic changes. Thus, a delusion that there were many people in the abdomen is explained by the sensations which arose from chronic abdominal distension; fears are explained by pressure on the thoracic organs.

VONDERAHE, Philadelphia.

SEVERE CHANGES IN THE NUCLEI OF THE GANGLION CELLS IN A CASE OF DEMENTIA PRAECOX. ARNOLD STOCKER, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **75**:47, 1922.

Stocker presents his discovery of marked changes in the ganglion cells of the brain from a case diagnosed as dementia praecox. The brain was from a woman aged 35 years, who was married, and the mother of three living children aged 8, 9 and 10 years, respectively. Five or six years before death she had a very difficult confinement and gave birth to twins; one of them was stillborn, the other died shortly after birth. The woman was ill for eight weeks at that time. In July, 1918, fifteen months before her death, the relatives noticed a change in her character. She angered easily, tired with her household duties and wished to be alone. Her speech was often inappropriate. At times she did not sleep and took hardly any nourishment. In this condition on several occasions she left home. At times she became very

angry and quite disturbed; at other times she was quiet and refused to talk. Finally she was committed to a hospital. At first she remained quietly in bed, kept her head under the covers, and only exceptionally made some small voluntary movement. She resisted the removal of the covers and buried her head in her breast. Memory and orientation were well preserved. After a time she became mute, and slept well, but it was difficult to feed her. Physical examination revealed cyanosis of the extremities and fleeting edema of the lower limbs. The reflexes were normal. She died in April, 1921, with symptoms of enteritis, but without fever. At necropsy the brain appeared normal macroscopically.

Histologically, however, the author found many cell changes. Examinations were made and descriptions are given of the following areas: the right and left second frontal; left first temporal; left and right caudate nucleus; left and right first frontal; left globus pallidus; left putamen; right thalamus; left subthalamic region; right locus niger; the medulla oblongata, and the cerebellum. In all these regions pathologic changes of considerable extent were found. Among the changes described, most weight is laid upon the presence of conglomerations of well stained, fine granules within the nuclei of the ganglion cells. In all regions examined, without exception, there were definite degrees of disorder of the ganglion cell protoplasm. As paradigms of the histological findings Stocker mentions the following: Among ganglion cells with high grade of chromatolysis, in which the cell protoplasm often stained very lightly and showed a vacuolated structure, neuronophagia and proliferation of the glia tissue were present. Many of the ganglion cells showed chromatophilia; often the cell structure was homogeneous so that cell protoplasm, nucleus and nucleolus could not be distinguished. Within the nucleus the granules already mentioned were deeply stained and of various sizes. Many of the ganglion cells contained considerable amounts of pigment as well as lipoid bodies. Pigmentation and lipoid bodies were also found in some of the glia cells and granule cells (*körnchenzellen*) of the vessel wall.

Ten cell pictures accompany the article. Stocker concludes by saying that he considers the ganglion cell which is described as an intermediate stage between the normal nuclear structure and the frequently described gray homogenization of the cell nuclei. He states that this finding is by no means pathognomonic for schizophrenia but that it is found in other pathologic brain processes; in fact he states that he has seen the same condition in epidemic encephalitis.

SOLOMON, Boston.

CLINICAL PICTURE AND PROGNOSIS OF THE FREQUENT MINOR
FITS OF CHILDREN. E. STIER, *Ztschr. f. d. ges. Neurol. u. Psychiat.*
80:143, 1922.

Pyknolepsy is the term used by the author to denote the convulsions which occur in infants or children, and which are not really epileptic in nature. He traces the subsequent history of a number of such cases in some detail and classifies them for prognosis according to the clinical manifestations. Certain characteristics, he says, will point one way or the other.

"The prognosis is absolutely good only when the attacks begin between the ages of 4 and 12. The children in question come only occasionally from families in which alcoholism, epilepsy or psychoses are present. The children themselves are not always free from intellectual backwardness, and occasionally have been subject to spasmophilia, to attacks of holding the breath or to tantrums. These features have no influence on the prognosis.

"The attacks commence out of a clear sky and without any previous excitement. The course is usually, but not always stereotyped until after months or even years, the attacks may cease abruptly, or much less often gradually, without leaving any residual nervous or psychic disturbance. Even with persistence of the attacks after puberty, the prognosis may be good.

"Symptomatically, the attacks are characterized by sudden and short interruption of the higher psychic processes, and by the monotony of the course. Enuresis, rigidity of pupils, slight raising of the arms, and slight, though not convulsive, rotation of the eyes and head are frequently present. On the other hand, deep loss of consciousness, injury in falling, running into objects, partial tonic or clonic convulsions, even when very mild, fear, palpitation or other vasomotor symptoms, and the occurrence of attacks during sleep, are always suspicious and are hardly consistent with a good prognosis.

"Onset of the attacks before the fifth year, fatigue or disorientation immediately after the attack, and control of the attacks by luminal are features which speak against the diagnosis of pyknolepsy and therefore against a good prognosis. It is unlikely that these attacks will resemble psychic fatigue or narcolepsy in the sense of Gélinau.

"On the whole we have in pyknolepsy a disease picture the etiology of which is quite obscure, but which presents a fairly definite symptom complex, and which can always be clearly differentiated from epilepsy."

FREEMAN, Philadelphia.

MANIFESTATIONS OF THE FEMALE CASTRATION COMPLEX.

KARL ABRAHAM, *Internat. J. of Psychoanal.* 3:1 (March) 1922.

Abraham summarizes and elaborates herein a frequently discussed but unclear concept of psychoanalysis. This concerns the wish of many women that they had been born male instead of female. He holds that this wish is manifested both in conscious and unconscious aspects and in multiform ways. The repressed envy of the male sex is shown particularly well in the dreams and symptoms of some psychoneurotic patients. Abraham first disposes of the rationalizations for the renunciation of the female sex, often offered by these women, such as deprivation of business and social privileges, disfranchisement, etc. He then declares that psychoanalysis has repeatedly discovered that, at certain stages of their development, little girls feel at a disadvantage as compared with boys because of their poverty in external genitals; they are envious of the penis. Although primarily without a feeling of inferiority, because she believes that all children are formed like herself, when the little girl learns the truth she may fabricate a theory that she too was once like a boy, but that, for reasons which are usually punitive, her penis has been taken away and has left a wound, the vulva. This theory is later reinforced by the flow of blood from the wound.

This discovery injures the girl's *amour propre*; it stimulates a sense of inferiority with a compensatory feeling of hostility, and impulse to rob. Abraham links with this the data of Freud's "Das Tabu der Virginität," the theory of the right of the first night, and the savage custom of defloration by a priest or friend. This feeling of envy and desire for revenge may be manifested in various ways, such as a marked interest in crippled men, in bald headed men and the like. It is compensated for symbolically in many women by the assumption of masculine habits and manners; in a less sublimated form it is replaced by an interest in the feces (overt and sublimated anal eroticism) because feces are parts of the body which are lost, and symbolize the penis.

Unadjusted repression of these so-called castration wishes may later lead to the dread of marriage, marital discord, frigidity and the well known phobias of wounds, knives and cutting.

Abraham illustrates his findings liberally, and introduces connotations which are not mentioned in this abstract. He concludes with a paragraph worthy of quotation, partly because of its intrinsic worth and partly because it takes up a matter very generally misunderstood by the antagonists of psychoanalysis: "To everyone of us who practices psychoanalysis the question occurs at times whether the trifling number of individuals to whom we can give assistance justifies the great expenditure of time, labor and patience. The answer to this question is contained in the above exposition. If we succeed in freeing such a person from the defects of her psychosexuality, i. e., from the burdens of her castration complex, then we obviate the neuroses of children to a great extent, and thus help the coming generation. (Because women whose ideas and feelings are influenced by the castration complex to an important degree, whether consciously or unconsciously, transplant the effects of this into their children.) Our psychoanalytic activity is a quiet and little recognized work, and for this reason all the more attacked, but its effect on and beyond the individual seems to us an aim worthy of much labor."

MENNINGER, Topeka, Kan.

THE TREATMENT OF GENERAL PARESIS BY INOCULATION OF MALARIA. WAGNER-JAUREGG, J. Nerv. & Ment. Dis. **55**:369 (May) 1922.

TREATMENT OF PARESIS BY INOCULATION WITH MALARIA. H. F. DELGADO, J. Nerv. & Ment. Dis. **55**:376 (May) 1922.

Wagner-Jauregg used a strain of malaria parasites obtained from the blood in a case which had not been treated with quinin, thus increasing the susceptibility to the drug when cure was desired. After one general paralytic had been inoculated, succeeding cases were inoculated by the injection of infected blood from the first case. Typical paroxysms of malaria appeared after an incubation period varying between six and thirty-six days. Eight or nine attacks were usually permitted to occur and the malaria was then checked by quinin; recovery occurred in all cases. Complete remission of symptoms of general paralysis occurred in all cases of short duration; remissions also took place in cases of early dementia, severe maniacal excitement with delusions of grandeur, and paralytic attacks. A course of neo-arsphenamin was given after the quinin therapy. No constant change was noted in the Wassermann reaction, globulin content, or cell count of the cerebrospinal fluid.

Delgado used the technic of Wagner-Jauregg, and reports four cases of general paralysis treated by malarial inoculation, as well as a case of probable dementia praecox or cerebral syphilis. This patient was accidentally infected with *plasmodium vivax*, and supplied the blood for the general paralytic cases. In two of the cases complete cure apparently took place; one patient improved greatly; one patient died—this was an advanced case with marked psychomotor agitation and with urinary and fecal incontinence. The author feels that there is ground for optimism in regard to the prognosis in general paralysis and calls attention to the views of Spielmeyer and Weichbrodt concerning the possibility of anatomic cerebral restitution. The author also notes that since ancient times, in one locality in Peru, the people treat leishmaniasis of the skin and mucous membranes by exposing themselves to malarial infection.

There is a tradition that, after a number of malarial attacks, the skin lesions heal. This is of interest because of the zoological relationship between leishmania and the spirochete of syphilis, and because in both infections arsphenamin is efficacious as a curative agent.

VONDERAHE, Philadelphia.

THE ETIOLOGY OF PHOBIAS. ENGLISH BAGBY, J. of Abnor. Psychol. **17**: 16 (April-June) 1922.

The writer presents two cases in which a phobia was removed by the discovery, in both cases accidental, of the etiologic psychic trauma. In the first case, a woman since childhood had been terrified at the sound of running water. This was eventually explained by an experience which occurred at the age of 7, when she seriously disobeyed an aunt; she ran away and became lodged in a wedge in the rocks beneath a water fall. All of this was concealed from her mother. The second case was that of a man who at the age of 55 discovered the cause of an abnormal terror of being grasped from behind. He traced it to an episode which occurred in the village grocery store, where, as a boy, he had been seized suddenly by the groceryman as he was stealing peanuts and had thereupon fallen screaming to the floor.

The writer frankly acknowledges that more thorough explanation could be made from the Freudian standpoint. He concedes that it would "be easy to see how a sex act would, in many cases, supply the element of guilt" but does not feel that in these two cases sex factors were present. He strangely omits to mention his indebtedness to Breuer, whose now little heard theories he tacitly supports or to concede that transference alone often yields rapid but temporary "cures."

MENNINGER, Topeka, Kan.

THE "NUQUO-MYDRIASIS" SIGN. E. FLATAU, Rev. Neurol. **28**:1200 (Dec.) 1921.

Flatau describes a new clinico-diagnostic sign—the mydriasis which follows neck flexion, and persists during the maintenance of this position and often for several seconds longer. This sign is present particularly in epidemic and tuberculous cerebrospinal meningitis in which cervical flexion is attended by pain, and the absence of sluggish pupillary reaction to light. It seems most marked in the acute and pre-agonal phases of such disorders, and in general seems to parallel other signs of meningismus, particularly the Kernig and Brudzinski signs. It has also been determined in serous meningitis following purulent otitis media, in hemorrhagic pachymeningitis, in third ventricle tumor with ventricular and sub-arachnoid hemorrhages, and in cervical spondylitis; but has not been determined in poliomyelitis with cerebral features, multiple sclerosis, tabes, uncomplicated intracranial neoplasms, epidemic encephalitis, functional disorders and non-neuraxial infections. In this respect it is of distinct value in differential diagnosis. As to the mechanism involved, the author finds this reflex to be: initiated through stimulation of the sensory endings in the field of the cervical vertebrae; transmitted along their fibers to the cervical cord; via the spinothalamic tracts to the ventral part of the thalamus and the hypothalamus; returned via the peduncles to the cervical cord; and distributed via the seventh and eighth cervical and first and second dorsal anterior roots to the superior cervical ganglions, and also over the first division of the trigeminus to the ciliary nerves, and finally to the dilatores pupillae.

RAPHAEL, Ann Arbor, Mich.

CERVICAL NEUROFIBROMA. EDWARD FLATAU and BRONISLAW SAWICKI, *L'Encéphale* **17**:617 (Dec.) 1922.

The case reported is that of a woman, aged 63, who for many years had an egg-sized, smooth, elastic and painless tumor in the left anterior triangle of the neck. It increased in size for a period of two years and the growth was accompanied by mild pains in the left side of the neck and down the left arm. There were no other symptoms and examination revealed no evidence of organic disease of the nervous system. At the necropsy it was found that the tumor originated near the posterior ganglion of the third cervical nerve. It extended outward through the intervertebral foramen, which was much enlarged, and ended distally in a cyst. The outer wall of the cyst was composed of nerve fibers; the inner wall was composed of connective tissue and had undergone cystic degeneration. The authors comment on the rarity of this condition and have collected only five similar cases from the literature.

They conclude from a study of their own case and the five previously reported that these tumors are evidenced first by the appearance of a swelling in the neck and, later, by symptoms of cord compression. Operation relieved the symptoms, and there was no instance of recurrence. The authors hold that these tumors originate in the proximal portion of the nerve and extend distally. In some instances they may originate within the spinal canal. The condition is not allied to Recklinghausen's disease; the nearest analog is the cerebellopontine angle tumor.

HYSLOP, New York.

TEMPERAMENT IN HARMONIOUS HUMAN RELATIONSHIP. M. J. REAM, *J. Abnor. Psychol.* **17**:58 (April-June) 1922.

"Rarely has the experimental method been used in attacking problems in human relationship." This work was carried out in a course of instruction at the School of Salesmanship of the Carnegie Institute of Technology. Ream's purpose was to answer the question with which he begins his article, "Do we prefer to work with people having temperaments similar to our own or . . . with contrasting temperaments?" He used a questionnaire method, and asked which of six given persons would be selected as a companion under certain conditions. These six, as well as the straw voters, had been previously grouped according to the Downey will-profile test as mobile (psychiatrists would understand better the term hypomanic and philosophers might prefer sanguine), or deliberate (phlegmatic). The conclusions rather strikingly indicated that the mobile types were more popular and that they prefer to work with persons of similar temperament, other things being equal. The selectors possessing a deliberate type of temperament were evenly divided in their preferences for companions.

MENNINGER, Topeka, Kan.

SEPTUM LUCIDUM TUMOR. SOUQUES, ALAJOUANINE and BERTRAND, *Rev. Neurol.* **29**:270 (March) 1922.

In the case reported, seemingly the first to appear in the literature, there was practically no anamnesis available, except an indefinite report of headaches, apparently of long standing. On examination the patient, a woman, aged 44, was found to be much deteriorated mentally in all fields, the reaction being essentially one of pronounced disorientation and confusion. Neurologically, there were noted: pupillary inequality with sluggishness in reaction to light, urinary incontinence, slight rigidity in leg-thigh flexion, slight lingual

tremor with complaint of severe unlocalized headache, and paresthesias of the lower extremities. Laboratory examinations, including examination of the spinal fluid, were quite negative. Examination of the ocular fundi was not carried out. The clinical course was one of rapidly progressing dementia, unattended by further neurologic developments, and death occurred after six months from pneumonia. The necropsy disclosed a glioma about the size of a hen's egg, with cystic and calcareous changes, which had developed primarily in the septum lucidum, and was associated with internal hydrocephalus and pressure effects upon the corpus callosum and cortex.

Comment is made upon the diagnostic difficulty presented by this case from the lack of localizing features; the outstanding reaction was merely a rapidly progressing dementia which, in this case as well as in others implicating the callosal field, the authors feel to be essentially of a secondary nature and dependent upon the associated, greatly increased, intracranial pressure.

RAPHAEL, Ann Arbor, Mich.

THE MENINGEAL SYMPTOMS OF ASCARIASIS. H. TURCAN, *Presse méd.* **30**:844 (Sept.) 1922.

The writer describes a typical case of ascariasis and calls especial attention to the differential diagnosis between this and tuberculous meningitis. Ascarids frequently cause general nervous phenomena: contractures, paralysis, and signs of meningeal irritation; less often they cause other phenomena. One case which simulated an acute mastoiditis is briefly noted. The pathologic change is still in dispute, but arguments are adduced against the theory of meningismus or pseudomeningitis, with reflexes caused by the direct irritation of nerve terminals in the intestines, and in favor of the theory of an actual toxic meningitis.

HUDDLESON, New York.

PLATO: A FORE-RUNNER OF PSYCHO-ANALYSIS. OTTO PFISTER, *Internat. J. of Psychoanal.* **3**:169 (June) 1922.

Pfister, a minister of Zurich, has made an effort to apply psychoanalysis in his professional work, and is a member of the International Association. This article attempts to point out that Plato held views which coincide with the theory of psychoanalysis (a thesis which was presented as early as 1915 by Nachmansohn). Pfister emphasizes Plato's eulogy of love, particularly physical love (which includes even homosexual love), and his unification of the love of God, the love of spouse, the love of child, and the love of mankind. Plato's conceptions of conflict are also considered parallel by Pfister and a few quotations will illustrate the points he emphasizes: "There are in the human body these two kinds of love; the desire of the healthy is one and the desire of the diseased is another. . . ." "The best physician is he who is able to separate fair love from foul, or to convert one into the other; and he who knows how to eradicate and how to implant love, whichever is required, and can reconcile the most hostile elements in the constitution and make them loving friends, is a skilful practitioner. . . ."

MENNINGER, Topeka, Kan.

THE STUDY OF VESTIBULAR NERVE FUNCTION IN MYXEDEMA. ROY A. BARLOW, *Am. J. M. Sc.* **164**:401 (Sept.) 1922.

Barlow notes from the work of previous observers that deafness is a frequent symptom in myxedema and also in cretinism. Fifteen cases of myxedema

were studied. Bárány tests revealed a retardation in response in 73 per cent. of the cases. Seven showed spontaneous nystagmus. In three cases there was lowered bone conduction. No spontaneous pointing fault was noted. The tests were accompanied by basal metabolic determination, but the changes in the ear function bore no apparent relationship to the metabolic rate. The clinical vestibular picture in myxedema, in the author's opinion, is definite and referable to delayed response to stimuli.

VONDERAHE, Philadelphia.

FAMILIAL INFANTILE BULBAR PALSY. E. D. PAULIAN, *Rev. Neurol.* **29**:275 (March) 1922.

Attention is directed to the apparent rarity of this condition. Only three authentic cases have been described thus far in the literature, the protocols of which the author discusses in full. On this basis, and his own experience, this disorder seems to have its onset most usually between the ages of 5 and 20. Paralysis (bilateral) of the superior division of the seventh cranial nerve appears to be the clinical feature which first becomes manifest, and is somewhat exceptionally accompanied by involvement of the middle and inferior divisions. As the syndrome develops, the tongue becomes atrophied and shows fibrillary tremor, and there are associated paresis of the pterygoid, masseter and palatine muscles, with corresponding disturbance in phonation, mastication and deglutition. Vagus and accessory paresis next present themselves and there are consequent respiratory difficulty and atrophic changes in the cervical musculature. The reflex field, however, is reported as essentially unchanged and there seems to be no indication of pyramidal disturbance. Death occurs after a relatively chronic course, either through inanition or aspiration pneumonia, or as a result of acute bulbar failure. Paulian comments upon the obscurity which obtains as to the essential etiology and pathology of this disorder. In connection with the latter, he mentions the possibilities of basic mitochondrial disturbance, ascending polioencephalitis and essential neuraxial agenesis, specifically of the cranial nerves.

RAPHAEL, Ann Arbor, Mich.

AORTITIS AND TRIGEMINAL NEURALGIA. J. MINET and R. LEGRAND, *Presse méd.* **30**:773 (Sept.) 1922.

Five cases are presented, which showed the "aortitis-trigeminal neuralgia syndrome." Central cerebral arteritis is thought to account for the neuralgia rather than sclerosis of the vasa nervorum. In one case, resection of the cervical sympathetic and of the first thoracic ganglion brought about an improvement in the aortitis. In other cases, improvement was obtained (at least of the neuralgia) by vasodilator and hypotensive medications or else by anti-syphilitic treatment.

HUDDLESON, New York.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Annual Meeting, Jan. 18, 1923

F. H. PACKARD, M.D., *President, in the Chair*

AN UNUSUAL CASE OF BRAIN TUMOR. DR. G. A. WATERMANN.

The patient, a man of 40, had been suffering from attacks of aphasia for fourteen years. The first year there were several epileptiform convulsions. During subsequent years there were attacks of aphasia, sometimes with nausea and sometimes with amnesia. He might go several days without these attacks; often they would occur many times during one day. There had been no headache, vomiting nor optic neuritis at any time in the course of the disease. The patient had suffered from attacks of ophthalmic migraine and also of asthma during his life in college; they had persisted every year since. When first seen by me he had been having jacksonian seizures for seven or eight years. These consisted of numbness that involved first the right foot, then extended up the body to the right cheek and ear, and finally down the right arm into the fingers; the numbness then passed off, the whole duration being one-half to one minute. These seizures followed the migraine attacks, with which were associated right-sided scotoma and right hemianopia.

In 1921 and 1922 the patient had a series of misfortunes: his father died, his mother had pneumonia, his boy had an accident that proved almost fatal, and much extra business responsibility was thrown upon him. Early in 1922, he began to have more and more lassitude, and the aphasia became almost constant, so that through the summer he found it difficult to converse and very difficult to understand what was said. The attacks of dizziness increased to twenty or thirty a day. He was unable to read although he still could play the piano and read music at sight. He found it difficult to understand simple questions and directions, and could scarcely speak a sentence without using wrong words or stopping to think of a word which he could not remember. In trying to read he would get two or three words very slowly and then be unable to go further.

The physical examination in October showed practically a normal condition. The pupils, optic nerves and retinas were normal; there was no nystagmus, and no change in the fields of vision, nor eye movements. Hearing was normal. The tongue was protruded straight without tremor. Grasp was normal in both hands. The wrist reflexes were absent. There was no ataxia, asynergy, adiadokokinesis, nor astereognosis. The knee-jerks were moderately active, and there was neither ankle clonus nor Babinski sign. The heart was normal with systolic blood pressure 120. The Romberg test was negative and he could stand comfortably on one foot indefinitely.

Through October and November the symptoms were sometimes better and sometimes worse. December 18, he suddenly became unconscious for a few minutes at the table. He was put to bed and throughout that day had increasing headache with almost constant vertigo, and seemed very sick. In the evening he vomited, fell into a deep sleep, and died shortly after midnight.

Postmortem examination revealed an extremely soft tumor, pinkish in color, that involved the whole temporal lobe from the tip to the posterior part below the angular gyrus. It occupied about one third of the substance of the lobe.

The interesting feature of this case is that the attacks of aphasia dated back for fourteen years, although persistent aphasia had existed only about six months. Microscopic examination revealed the fact that the tumor was undoubtedly of recent development, and that the earlier aphasic attacks and convulsions had been due to a degenerative process in the temporal lobe. The tumor was made up of rapidly growing cells as indicated by the presence here and there of mitotic figures; it was very vascular and contained many small hemorrhages. Scattered through the tumor were numerous masses of calcified corpora amylacea; but these bodies existed also in the uninvolved brain tissue and thus indicated a previous degenerative change in the temporal lobe which had subsequently been invaded by the tumor. It seems probable, therefore, that the earlier attacks of aphasia were due to a degenerative lesion of the temporal lobe, and that the latter, intensified symptoms were due to a glioma in this region, that perhaps developed in some indirect manner as the result of the intense mental strain to which the patient had been subjected.

DISCUSSION

DR. G. L. WALTON: The duration of efficiency in spite of the gradual growth of a tumor of this variety gives the case a special interest. There can be no question that the tumor was the cause of the symptoms present when I saw him in 1909, though I have no recollection of the process of reasoning by which I reached the diagnosis. Indeed, until reminded of it by Dr. Waterman (and by Dr. Clap, to whom I sent him for ocular examination) I did not remember that I had done so. My notes, however, would indicate that epilepsy was made improbable by the prominence of gastric symptoms, hemianopia, and aphasia at the time of the attacks, as well as by the persistence of aphasia after consciousness was recovered; that migraine was excluded by the loss of consciousness and lapses of memory, as well as by the aphasia; this last would have been only fleeting and premonitory, in the usual event that it were a symptom of this disorder. As in this case, the question whether long standing migrainous attacks were connected with the new growth or merely coincidental, came up in the case of Dr. D. that many of us remember; he died from pituitary tumor at the age of 28, but had been subject to attacks of migraine, preceded by hemianopia, for a great many years. In that case, however, the hemianopia became permanent during the last year of his life, which seemed to show that the "sick headaches" were other than a coincidence.

DR. W. J. MIXTER: I feel that this case has a real lesson, namely, that when new growth is suspected, operation is indicated even in the absence of choked disk or other evidence of general pressure. Focal epilepsy is frequently an indication of a local process involving the cortex, and as such requires exploration. We are all of us too prone to await the onset of general pressure symptoms before advising operation. The neurologist should remember that the difficulties and dangers of exploratory craniotomy are remarkably increased by increased intracranial pressure.

I am perhaps a radical, but I am strongly of the opinion that cases of focal epilepsy should always be explored. Operation in Dr. Waterman's case could have been palliative only, but to my mind would have been indicated, especially as I thought from the history that the lesion was probably an

arachnoid fibroma or a localized collection of fluid beneath the arachnoid rather than a glioma. An osteoplastic craniotomy, such as would be performed in a case of this kind, should expose the motor cortex from the lower edge of the leg area down, and also most of the temporal and part of the frontal lobes.

BARBITAL INTOXICATION. DR. A. WARREN STEARNS.

During the past five years certain cases have come to my attention, a unique feature of which is that a single large dose of barbitol has caused a state of intoxication, either pleasurable or a relief from unpleasant symptoms, that lasted from twenty-four hours to several days. The evidence does not warrant an opinion as to whether or not this is a peculiar reaction of certain people to barbitol.

CASE 1.—A man in the sixties, who had been a periodical drinker for many years and had used barbitol extensively during the sobering-up period, was found one day in a state of profound intoxication. Movements of the hands were clumsy, the speech was thick, the eyelids heavy, and the reflexes essentially negative. He was mentally clear but thought processes, though accurate, were very slow. He was also somewhat jocose. This state lasted for two days. The patient was seen in several subsequent attacks. In at least one, there was assurance that the intoxication was due to some substance taken before the attack, the effect of which lasted for at least twenty-four hours. It was later learned that this man had taken large doses of barbitol; the exact amount was not determined.

CASE 2.—A trained nurse in the thirties, who had necrosis in the jaw with a great deal of neuralgic pain and insomnia, was found one day in a state of intoxication which at that time was believed to be alcoholic. Subsequent investigation satisfactorily ruled out alcohol. I first saw her in the evening; she had returned home late in the afternoon in an intoxicated condition: very unsteady, speech thick, eyelids heavy. The reflexes were unaltered. It was supposed that she had taken some drug, so her clothes were taken away and she was closely observed. She slept a little but for the most part remained in this condition for two days. I saw her in several subsequent attacks without knowing the cause of the condition until finally her brother saw her take the whole contents of a tube of barbitol tablets containing 100 grains. The intoxication from this dose lasted about forty-eight hours.

CASE 3.—A man in the forties, a chronic alcohol addict, was found in a condition very similar to the above but with no alcoholic odor on his breath. He admitted that for the past year he had been taking barbitol in doses of from 60 to 75 grains, that produced an intoxication lasting from twenty-four to forty-eight hours.

CASE 4.—A man in the late forties, who had had repeated operations for gastric ulcer and had complained of great pain, was found unconscious one day and was supposed to have attempted suicide. After twenty-four hours he woke comparatively clear. He has since been observed in several attacks like those already described, and has admitted taking from 20 to 40 grains of barbitol for the purpose of producing this state.

CASE 5.—A man in the forties complaining of gastric distress and insomnia, had taken barbitol in an attempt to get relief. While on the train with his brother, he took the contents of a tube of barbitol tablets; the exact number is not known. When seen six hours later, he was clumsy, unsteady, and the

eyelids drooped so as to obscure vision; the knee-jerks were absent. The next morning when I saw him he was in the same condition except that the reflexes were all active. The following morning he still showed symptoms, but they were less marked. He admitted taking large doses of barbital in order to get relief from his symptoms.

DISCUSSION

DR. F. J. FARNELL: In several cases of barbital as well as phenobarbital poisoning seen by me during the last few years, all have presented absent superficial reflexes.

DR. F. H. PACKARD: While I have no definite memory of the details of the cases, I remember that many years ago Dr. Tuttle told me of the possibility of poisoning by barbital; he had seen a number of cases. In those cases, as I remember it, barbital was not taken for the purposes reported by Dr. Stearns; the poisoning was accidental. Barbital was a comparatively new drug at the time and as veronal was advertised as harmless, in contrast to some other hypnotics, poisoning resulted from taking large doses when small doses had not produced the desired results. Poisoning by bromid, which Dr. Walton has mentioned, used to be rather commonly seen at the McLean Hospital; many patients arrived in a state of delirium, much exhausted physically, with badly coated tongue and sordes on the teeth. There were frequently paraphasia and aphasia, and a bromid eruption was usually to be noticed. These were very gratifying cases to care for, as with withdrawal of drugs, proper catharsis and feeding, they very speedily recovered. The usual history was that because of nervousness and sleeplessness the family physician had begun by giving small doses of bromid, which had been increased to large doses in an attempt to produce the desired effect, and had been continued regularly for some time. As a rule the history indicated that little food had been taken for some days. I have seen many such cases outside in consultation, where a removal of the bromid and feeding soon resulted in the patient's recovery.

THE PSYCHIATRIC IMPORTANCE OF THE KUHLMANN TESTS.

DR. JAMES S. PLANT.

A new age-level set of psychological tests, which were standardized largely upon institutional cases, is presented. The present study deals with results in the unselected and varied material that comes to the Judge Baker Foundation. These tests stress mental control. They have the following advantages over the Terman tests: (1) being new there is less likelihood of previous coaching; (2) the responses are far less open to interpretation; (3) they are largely performance tests; and (4) they involve language to a much smaller extent. They have the following disadvantages: (1) they require a little longer time and demand much more sustained attention, and (2) they are very hard to motivate.

Forty-five unselected cases have been used. Eight of these were mildly psychotic or "pre-psychotic." Psychiatric diagnoses were made by other physicians without knowledge of the test results. All cases were also given a wide range of other tests.

Normal cases showed a correlation between Kuhlmann and Terman tests of 0.95 with a probable error of ± 0.01 . With one exception the "psychotic" cases showed much lower Kuhlmann than Terman intelligence quotients. The least difference between the Terman and the Kuhlmann intelligence quotients

for psychotic cases was as large as the largest difference for any normal case. In one psychotic case the Kuhlmann intelligence quotient was 26 points lower than the corresponding Terman.

"Mental control" is largely dependent upon the affective life. Psychologic tests have almost entirely busied themselves with intellectual functions. It is a matter of great psychiatric importance that a set of *standardized* tests have touched, however roughly, the emotional field.

F. L. WELLES, PH.D.: It is not clear to me just how Dr. Plant regards mental control and emotion, but it seems that he looks upon them as very close together, in that great fluctuations of a test dependent on mental control indicate instability of emotional life. The balance of probability is on this side, but it is doubtful if the Kuhlmann tests differ in this respect from a considerable number of standardized performance tests, such as those worked out by Pintner and Patterson. In this respect the Kuhlmann tests do what is done by any test involving continuous effort, of which there are many. Lesser correlation in psychotic, as compared with normal, material probably exists between any two tests. The Stanford scale correlates with itself, so to speak, least in psychotic material. It has been found most regular with normals, less so with feeble-minded, still less with psychotics in general, and least regular of all in organic or toxic conditions. In general, new scales nowadays add little in the way of more accurate methods of measuring intelligence, though some, like the Myers Mental Measure, make advances in convenience and practical serviceability. New test series are advantageous to have, however, as alternates to old tests with which the patient has become familiar. The Kuhlmann tests are a welcome addition to psychometric resources, but special distinction claimed for them is perhaps open to question.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 19, 1923

H. I. DAVIS, M.D., *President, in the Chair*

PARALYSIS AGITANS IN A GIRL OF TWELVE. DR. W. G. STEARNS.

In this case the typical tremor of Parkinson's disease is present in the hands, arms and shoulders of both sides. The patient feeds herself with great difficulty. She can write legibly but only by pinning the forearm of the right hand beneath the weight of the body. The upper extremities lack markedly in muscular power when compared with other muscles of the body. There is only a slight amount of rigidity in the affected muscles, but the characteristic, slightly semiflexed condition is present. The muscles of the head and neck are not affected. The muscles of the trunk, except those about the shoulders and those of the lower extremities, remain unaffected. There is no tremor during sleep. There are no disturbances of sensation and the reflexes are normal.

In early childhood this patient had chickenpox, mumps and measles. At 6 years of age she "fell down the cellar door," but was apparently uninjured. She was an active robust child. At the age of 12 the tremor first appeared, equally in the two hands; it has very gradually increased in amplitude and has slowly extended to involve the hands, arms and shoulders. At 13, men-

stration was established normally. At 14, she had her first sore throat. At the present time the patient is 16 years of age and is, except for the condition of the arms, in good health, strength and spirits. She is in the third year at high school and appears normal mentally.

CYCLOPLEGIA IN A GIRL OF TWELVE. DR. W. G. STEARNS.

This patient, a girl aged 10, complained that near vision was blurred, and was unable to read even the very largest newspaper headlines. She was hypersensitive, irritable, and unable to concentrate her attention. In early childhood she had measles, whooping cough and chickenpox, all in mild form, and has never had any serious illness.

Nov. 16, 1922, she had fever at night, and her throat was sore with gray spots on it. Her mother treated the throat with tincture of iodine and a mouth wash. She promptly recovered and was out of school for only one week. December 3, seventeen days later, she could not read. December 19, about five weeks after the onset, they first consulted an oculist, who found complete paralysis of accommodation. She had 2.25 diopters refraction error and was given a corresponding lens; this enabled her to read at 16 inches, but no less. The pupils were not dilated, and reacted well to light; the eyes were otherwise normal. Weekly visits were made thereafter, and December 26, forty days later, the oculist's findings were the same. She was then referred for an opinion as to the pathogenesis. She had at that time a slight nasal twang to her voice, which suggested paresis of the soft palate. January 2, the condition was much the same but the nasal quality of tone had disappeared. January 16, the pupils were normal, reacted promptly to accommodation as well as to light, and vision was normal.

There can be no doubt that on November 16 the child had diphtheria. Two and a half weeks after the diphtheria, disturbance of vision appeared. At the first examination, the oculist found complete loss of accommodation with normal reaction to light. My excuse for presenting this case is that there was such clear-cut, well defined and complete loss of accommodation, in spite of which the pupils behaved normally in every other way.

DISCUSSION

DR. SANGER BROWN: What was the condition of the deep reflexes when the cycloplegia was at its height?

DR. PETER BASSOE: I would like to ask if there is any possibility that the first patient had a mild attack of epidemic encephalitis. We do see paralysis agitans-like syndromes following encephalitis of the ambulatory type. I saw such a case in the dispensary last week in a young man who gave a history of having had a mild indisposition in October, 1919. He saw double for a couple of days and stayed away from work one day only, but I believe, in view of his present condition, that this was enough to justify a diagnosis of encephalitis. True paralysis agitans in a child of 12 is extremely unusual.

DR. HUGH T. PATRICK: Did you consider the possibility of Wilson's disease or pseudosclerosis in the first case?

DR. W. G. STEARNS: In the second case the deep reflexes, including the knee-jerks, were slightly increased. The patient was easily irritated and hypersensitive.

In the first case I could get no history of any untoward events in the child's life except her falling "down the cellar door" when between 6 and 7 years of age. She had not menstruated at the time of this trouble and had never had a sore throat until she was 14. I had not considered Wilson's disease.

A CASE OF CONCUSSION OF THE SPINAL CORD WITH A CLINICAL PICTURE OF AMYOTROPHIC LATERAL SCLEROSIS.
DR. G. B. HASSIN.

This article will appear in full in a later issue.

HYSTERICAL AMNESIA FOLLOWING PHYSICAL INJURY—A CASE STUDY. DR. CHARLES F. READ.

The patient, a man, aged 26, with a somewhat neurotic background, suffered a slight injury while working in a coal mine, Oct. 11, 1922. He was struck over the right shoulder blade by a falling piece of coal a little larger than two fists; he did not become unconscious but complained of being dizzy and soon began to act queerly. He was taken to a hospital where he exhibited some hysterical behavior; following this he came to himself with an amnesia which extended back to Jan. 7, 1914.

Inquiry revealed the fact that prior to the latter date he had become involved in an affair at school with a married woman, and, after a quarrel with a friend who ventured to advise him in the matter, the patient "lost himself" for a period of several days; following this he "came to" in a mining town hospital in October, 1922. Aside from some vague dream-like recollections, entirely disoriented as to time and place, the amnesia was complete when the writer saw him some weeks after the accident. Simple inquiry failed to accomplish anything by way of free association so, inasmuch as the time was short, the patient was hypnotized and the memories of the intervening years were readily recovered. A day or two later in the waking state the patient succeeded in recovering these memories and was soon able to leave the hospital.

The case is of interest, because, although the amnesia followed physical injury, it was entirely of a hysterical character and was very probably occasioned by the fact that the patient, naturally of an artistic sensitive makeup, and with a history of a previous flight when a youngster, had gone to work in the mines because of a sense of duty toward a tuberculous sister. Two uncles had been killed in the mines and two brothers injured; the thought of the mines disgusted him, but in spite of this, in order to help the sister, he mined coal during the day, while for his own pleasure he wrote upon a novel at night. The conflict between a sense of duty and the desire for artistic creation gave rise to the final dissociation, which was precipitated by the slight accident in the mine.

Recovery was symptomatic and should be followed by psychoanalysis until the patient becomes acquainted with his own personal makeup and learns how to deal with it; else there will very possibly be other hysterical flights of one kind or another in the future.

DISCUSSION

DR. CLARENCE E. NEYMANN: I would like to ask Dr. Read if he had an opportunity to go into this patient's period of conflict at the age of 17. It seems to me that in his amnesia he gets out of one conflict, coal, dust and grime, and throws himself headlong into a sexual conflict. Did he find any

enjoyment in the comparison between the fairy-like creature and the barbaric beauty? Is there not behind that some lack of sexual satisfaction in the intervening years, and is he not the sort of person who always wants a sort of pot-pourri, the heavenly and earthly love? If he is that sort of person, is this not the cause of the amnesia, rather than the fight with the dirt and grime of poverty? Would that not explain why he included the army period, because in this army work he had no chance to enjoy the heavenly and earthly love which he evidently desired?

DR. RALPH C. HAMILL: I have been working on a case of amnesia for some time in a young man who stole, on two different occasions, and then went into periods of amnesia; the first one lasted from the time he got out of the army until his stealing was discovered, which covered a period of, as I recall it, a little over a year. With him, as with this patient of Dr. Read's, it was very interesting to note the emphasis which he put on his fear of his father's finding out about it and the punishment that would be meted out to him by his father. Then it was interesting that the first person from whom he stole reminded him very strongly of his father. Unfortunately, he got away from me before I managed to connect up, as satisfactorily as I think could have been done, the relation between stealing from this man who resembled his father and early, youthful experiences; but there was the very strong feeling of fear in his mind, especially of punishment being meted out to him by his father.

DR. CHARLES F. READ: I do not know just what to say in response to Dr. Neymann's question. The patient evidently did get some considerable enjoyment in writing out the story of his attachment to these two women; it is, perhaps, not unusual in the male sex, to take pride in their amours. Just what relationship this bears to his amnesia is problematic. I did not get back at all into his very early life and therefore do not know what his personal makeup is, as I acknowledged. I merely secured a symptomatic recovery. Evidently he had learned how to become amnesic and, when the second crisis came, snapped back through the intervening years, through his period of artificial war-time sublimation, back into the days preceding the first amnesia.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 26, 1923

CHARLES H. FRAZIER, M.D., *President, in the Chair*

TELANGIECTASIS OF THE SPINAL CORD. DRs. WILLIAM G. SPILLER,
and CHARLES H. FRAZIER.

This report will be published in full in a later issue of the ARCHIVES.

A MENINGIOMA BOTH INTRADURAL AND EXTRADURAL,
REMOVED FROM THE SPINAL CORD WITH COMPLETE
RECOVERY. DR. WILLIAM G. SPILLER.

History.—The patient, a woman 20 years of age, began to be weak in the lower limbs in April, 1922, having been entirely well until this time. She fell several times during April and on one occasion slipped to the bottom of

the stairs but was not injured. She had occasionally during May some stiffness and numbness in the lower limbs, but these were not persistent. Her gait was a little incoordinate. The little finger of each hand began to be numb. On July 19, the right patellar reflex was about normal and the left a little exaggerated. The right Achilles reflex was not obtained and the left was about normal. The Babinski reflex was not typical, for while the slightest irritation of the sole of either foot caused the big toe to assume a position of persistent overextension, the position may have been the result of voluntary movement. She was able to walk with very little incoordination. She had no distinct impairment of objective sensation.

The patient had had much mental disturbance, family affairs had excited her, and as she undoubtedly improved under psychotherapy, it was impossible to exclude at that time a psychic complication. It seemed best to have her go away to the seashore for a short time with the understanding that if she should get worse she would return for observation. It would have been unwise to recommend operation until the symptoms became more pronounced and the patient would not have accepted operation at that time. She did not return from the shore until late in the summer, and did not agree to have an operation until October. Her condition then was much worse and was as follows:

Heat, cold and pain sensations were impaired in the lower limbs and trunk as high as the second intercostal space on each side, and on the inner side of each upper limb as far as the hand. Tactile and vibration sensations were not impaired. The patient stood with the greatest difficulty, even when supported, but if supported she could take a few steps with the right heel fully 2 to 3 inches from the ground and this heel could not be brought to the ground, whereas the left heel could be placed on the ground. The right leg could not be fully extended on the thigh on account of contracture of the flexors of the legs. The spasticity of the lower limbs was excessive and interfered with obtaining the tendon reflexes, but occasionally when it was less the patellar reflexes could be obtained and were exaggerated. The feet were in extension and spasticity prevented ankle clonus. A very typical Babinski reflex was present on each side.

The little and ring finger of each side were partially flexed at the phalangeal articulations, especially the little finger, and the muscles of each hand were somewhat atrophied. The biceps and triceps reflexes were lost or nearly lost on each side. There was no ocular sympathetic paralysis.

The signs all pointed to a lesion having its upper level about the eighth cervical segment, and affecting the lateral and anterolateral portion of each side of the spinal cord. The posterior columns and posterior roots evidently had escaped, for there had not been any pain at any time except the discomfort caused by the spasticity. The right side of the cord seemed to be a little more affected.

The diagnosis made before the operation was tumor on the anterior part of the spinal cord at the eighth cervical segment.

Operation.—Nov. 1, 1922, the laminae of the fifth, sixth and seventh cervical and the first thoracic vertebrae were removed. On a line beneath the lamina of the sixth cervical, there appeared on the right side of the cord what was thought at first to be an extradural tumor. In rongeur-ing away the lamina toward the right, it was found that this tumor extended a distance of 1 or 2 cm. into a bony cavity about 2.5 cm. long, 2.5 cm. deep and 4 cm. across from

the lamina on the left side to the bony wall of the cavity on the right. In attempting to liberate the tumor from the dura it appeared to be firmly attached at one point and on closer inspection was found to continue within the dural sac. A T-shaped incision was then made in the dura and a small intradural tumor lying on the anterolateral aspect of the cord was found. It was free from all attachments and readily enucleated. The larger growth, however, was very firmly attached at its base, which appeared to be at the outer side of the bony cavity, so that in its removal portion of the capsule was stripped from the tumor. It is difficult to determine where the tumor originated. The intradural tumor had made but little impression on the cord and the larger extradural tumor, because of the size of the cavity in which it lay to the right, caused little pressure on the cord. None of the roots passed over the extradural portion of the tumor.

For several days after the operation the spasticity and involuntary jerking of the lower limbs were excessive and caused the patient great pain, but this gradually ceased. The lower limbs regained their normal power, the impairment of heat, cold and pain sensations in the lower limbs disappeared and the patient now has little or no symptom caused by the tumor.

Pathology.—Examination of the tumor showed it to be an endothelioma, or possibly a fibroma. It is sometimes difficult to distinguish between these two forms of tumor occurring in the central nervous system as sections from one part may resemble a fibroma and from another part of the same tumor may have the appearance of endothelioma. Perhaps one may best solve the difficulty by employing the noncommittal name of meningioma suggested by Cushing.

It would seem that the tumor had its origin probably from the place of exit of the spinal roots from the vertebral canal, and that it gradually wore away the surrounding bone as it grew, making in this way a nest for itself, and thereby causing no pressure upon the spinal cord. The symptoms were evidently caused by the intradural tumor, which exerted pressure on the anterior and lateral parts of the spinal cord. It is exceptional that a tumor should be partly intradural and partly extradural.

DISCUSSION

DR. CHARLES H. FRAZIER: The intradural tumor was elongated and of the size of the average extramedullary intradural endothelioma. Through a small opening the intradural tumor communicated with what I believed to be the primary extradural growth.

AN ENCHONDROMA OF THE BRAIN REMOVED WITH RECOVERY.

DR. WILLIAM G. SPILLER.

History.—D. D., 45 years of age, was admitted to the hospital, May 16, 1922. On Sept. 14, 1918, he was on a train to which was attached a car containing soldiers who had been injured, and he was much affected by what he saw. After awakening on September 16, and while still in bed, he had a convulsion in the right lower limb. It is uncertain whether his right upper limb was implicated, and after the convulsion he was numb on the right side of his body. A physician who saw him while he was in the convulsion diagnosed the condition as hysteria. He had a similar attack in the right lower limb about a month later. The attacks occurred at long intervals until about January, 1921, when they came about once a week. February 3, he had two severe attacks

within one hour, and since then he has been lame in the right foot. His ankle and knee on the right side were affected and his gait became abnormal. In April of this year he had an attack which implicated the right upper limb as well as the right lower limb, and for a time after this attack his right upper limb was powerless, but although he regained much use of this limb he had not been able to write well with the right hand. During the six weeks preceding his admission to the hospital he made many mistakes in calculations, which was striking as he was a bond salesman. He had some difficulty in remembering names and, in reading, he was obliged to read some sentences more than once in order to get the meaning. He had had occipital headache about once a month for years.

Dr. de Schweinitz found the eyegrounds normal. The innervation of the right side of the face in the lower part was a little impaired. Sense of position was impaired in the right hand and this hand showed *adiadokokinesia*. The biceps and triceps tendon reflexes were a little prompter on the right side, but there was no decided weakness of the upper limb. The right patellar reflex was a little exaggerated. Ankle clonus was pronounced on the right side only, and the Babinski sign was positive only on the right side. The sense of position was distinctly impaired in the right big toe. The right foot turned in and the man walked on the outer border of this foot, and dragged his right toes.

He seemed to have beginning word deafness and word blindness, but these disturbances were slight. His inability at times to get the word he wanted, although he understood all said to him, with some difficulty in reading, were suggestive of early word deafness and word blindness.

The diagnosis was made of a lesion, probably tumor, that irritated especially the center for the right lower limb, but also the center for the upper limb, and implicated the parietal lobe, thus causing, possibly by pressure, slight disturbance of the left angular gyrus and left first temporal convolution. The tumor was believed to be in the motor and parietal region, and as convulsive movement of the lower limb was for a long time the only sign, the tumor was believed to be cortical or near the cortex, especially near the paracentral lobule. Laboratory tests of blood and spinal fluid for syphilis were negative.

A roentgenogram, taken by Dr. Pancoast, showed very distinctly a tumor in the suspected region. The patient was referred to Dr. Frazier for operation, which was performed June 3, 1921.

Operation.—A flap was reflected to uncover the motor area of the left hemisphere. It was noted that the bone was unusually thick at all points and particularly so in the temporal region. On reflection of the flap the tumor at once presented itself in the upper third of the opening. It was hard, fixed, irregular, and adherent to the falx, but was sharply defined and easily separated from the surrounding hemisphere although at several points, venous channels bridging across from the hemisphere to the tumor, had to be tied and cut. The point of origin of the growth, or at least the point of greatest attachment, was an area from 2 to 3 cm. long and 1 cm. wide along the falx at the level of the longitudinal sinus.

In separating the tumor at this point there was sharp bleeding which was readily controlled by tampons. At the conclusion of the operation every attempt to remove these tampons was followed by recurrent bleeding. A fascial transplant at once controlled this. At the conclusion of the operation there was no bleeding from the site of the tumor, but there was from the space between the

dura and the bone. This was controlled eventually by suturing the dura to the galea, thus securing compression of the dura against the overlying bone.

While the wound was being closed the patient was given 600 c.c. of blood from a suitable donor with 500 c.c. of salt solution. The transfusion was divided into two stages, the first portion being given when the wound was about to be closed and the second toward the end of the operation. The wound was closed without drainage.

The pathologic diagnosis was enchondroma.

The patient made a satisfactory recovery and was seen in September, 1922. At that time his condition was about normal except that he was still a little lame in the right foot.

PRESENTATION OF A CASE IN WHICH A PRESUMPTIVE DIAGNOSIS OF TUMOR HAD BEEN MADE THREE YEARS BEFORE.

DR. CHARLES H. FRAZIER.

This case is presented because of the entire freedom from symptoms of intracranial pressure after a period of over three years. A diagnosis presumptive of tumor was made and decompression performed Nov. 28, 1919.

M. F., aged 25, was referred to the neurosurgical service of the University Hospital by Drs. William G. Spiller and Charles Schoff. She has three brothers, all of whom are regarded as "backward," but the patient is quite normal, having graduated from high school and college. In November, 1918, she had a severe attack of influenza after which she had headache that gradually became worse, until in May, 1920, she was admitted for observation to the hospital. The family said they were afraid she might hurt herself as she wanted to press her head and even butt it against the wall. At times her physician said she was quite "flighty" and she suffered from insomnia. She complained of ringing in the right ear, inability to open the right eye as well as the left, and dizzy spells.

On admission to the University Hospital she was examined by Drs. Spiller and de Schweinitz. The positive findings were very few: the lower part of her face was a little coarse suggesting early acromegaly; the tongue was protruded slightly to the right but there were no other cranial nerve palsies. The patellar reflexes were increased, but there was no Romberg sign nor disturbance of gait, and no incoordination. There was a double choked disk of from 4 to 5 diopters with fresh hemorrhages in the right eye; vision in the left eye was two-fifths. Concentric contraction of visual fields was found. The findings suggested intracranial pressure, possibly due to sinus infection.

The subsequent course of events excludes the possibility of tumor, but at the operating table I realized that the symptoms were not due to a neoplasm. When there is a large collection of fluid in the subdural or subarachnoid space, the free escape of which at the operation relieves the pressure at once, one is quite justified in assuming that the pressure phenomena are not of tumor origin. In the absence of a tumor we must assume a lesion of inflammatory or infectious nature, determining a serous meningitis. That this may follow influenza is quite conceivable and my corrected diagnosis in this case is serous meningitis.

The advantages of subtemporal decompression in the presence of a high grade papilledema of doubtful origin could not be illustrated more strikingly than in this case. Dr. de Schweinitz observed that he had never seen a more rapid subsidence of a choked disk. Much has been said of late to discredit the decompressive operation, but it still has a field in properly selected cases and it is in doubtful cases awaiting final diagnosis that the operation should prove of greatest service.

THE ADMINISTRATION OF HYPERTONIC SALT SOLUTIONS FOR THE RELIEF OF INTRACRANIAL PRESSURE. DR. TEMPLE FAY.

The management of intracranial pressure following trauma or obstruction of the ventricular system by new growths, has offered grave difficulties to the neurosurgeon. The recent work by Weed and McKibbin with hypertonic salt solutions has demonstrated that the intracranial pressure may be rapidly reduced by dehydration, the fluid of the ventricles being reabsorbed into the blood to maintain an isotonic solution.

If 50 to 100 c.c. of 15 per cent. sodium chlorid solution is injected into the veins of a patient suffering from increased intracranial pressure, there will be a decided relief of symptoms within twenty minutes. There will also be a decided reduction of tension and volume of the brain substance. This has been clearly shown in cases where the brain was exposed for exploration and the dura was found to be too tense to warrant opening. Seventeen per cent. of the craniotomies performed by the neurosurgical service at the University Hospital received this salt solution by vein, in order to assist in safely opening and closing the dura in cases with a great amount of pressure. In all but one case it reduced the pressure so that exploration was possible in ten to twenty minutes.

The administration of salt solutions by mouth is somewhat slower and yet quite as effectual. Sodium chlorid is often difficult to take and the patient does not retain it well. Thirty c.c. of a 30 per cent. solution may be used. Cushing and Foley found the brain volume reduced 50 c.c. in one case of dilated ventricles.

The use of magnesium sulphate solution by mouth or rectum has been found in this clinic to be of the greatest value, and very rapidly effectual in all cases of increased intracranial tension. As a preoperative measure it is used as a routine to relieve tension and facilitate exploration. Post-operatively in cases of so-called "medullary edema" where the pulse and the respiration begin to fall rapidly, an ounce and a half of magnesium sulphate crystals by mouth in a glass of water, or 3 ounces of crystals in 6 ounces of water by rectum, will cause a return of the respiration and pulse to normal and relieve the depressed state of the lower centers.

The use of magnesium sulphate in treating all forms of increased intracranial pressure has been very gratifying in our experience of over two years. It will relieve the headache, vomiting and choked disk of the early stages of increased pressure and a patient can be made comfortable until operative intervention can be obtained. It assists in the localization of tumors by removing the pressure symptoms that may mask or exaggerate those of localizing value. In cases of coma and medullary depression when increased intracranial tension was the cause we have seen it bring marked relief. In accident cases with intracranial injury or fracture of the skull followed by the picture of "medullary edema" it is of great value in checking the fall in pulse and respiration, and should be given as a routine, preferably by rectum.

The advantage of magnesium sulphate over saline solution is apparent. It is not absorbed into the blood, as is the sodium chlorid solution, but exerts its action as a hydrogog, rapidly withdrawing fluid from the blood stream into the intestinal tract. This causes concentration of the blood plasma and there is a call upon the tissue fluids to meet the deficit. The ventricular system is very generous in its contributions and pressure in the spinal system falls rapidly. Magnesium sulphate is easy to give and the patients retain it well.

Sodium chlorid on the other hand when given by mouth or vein causes a hypertonic condition of the blood, which draws fluids from the tissues until the entire body is isotonic. Here then after a slight period of reduced pressure there is a secondary salt retention and edema that follows from the excess of the substance in the body. Sodium chlorid, given by mouth, is very irritating and frequently causes nausea and vomiting; even when administered in salol-coated capsules patients complain bitterly.

DISCUSSION

DR. CHARLES H. FRAZIER: The use of magnesium sulphate solution as a substitute for sodium chlorid is, as far as I know, original with Dr. Fay. He has made a very important contribution to the management of intracranial pressure when associated with brain tumors. Not only has magnesium sulphate proved effective when the pressure was due to largely dilated ventricles, but even in the absence of these, and in this particular it may prove of greater value than sodium chlorid. The administration of the latter by mouth has not proved feasible with us, it being most effective when given intravenously. How often one may repeat these injections safely we do not know. Hence to have a substitute in magnesium sulphate, which can be given by mouth or by rectum, and be frequently repeated with perfect propriety, is a matter, I think, of considerable moment.

CASE OF ENDOTHELIOMA OF BRAIN ONE YEAR AFTER OPERATION. DR. WALTER FREEMAN.

This patient, who had previously been in good health complained of inability to use his right hand since 1919, when he noticed twitching of the right side of his face, recurring at intervals of two or three weeks; it was later associated with loss of consciousness. These twitching movements were repeated often, and were accompanied by numbness and tingling in the right hand. Later this hand became clumsy in its movements, with slight weakness; he had to direct it by sight, and was unable to tell the denomination of coins in his pocket. Recently the patient noticed drooping of the right corner of the mouth. In walking he dragged the right leg. There were no involuntary movements of the arm or leg. He had some difficulty in talking, being unable to find the right word. In writing, the characters were formed with great difficulty. Headaches had been paroxysmal, nocturnal and very severe, but were not associated with vomiting. There was no diplopia although the eyesight was becoming poor.

Physical examination revealed drooping of the right side of the mouth with weakness of the right lower facial segment. Power was diminished in the right hand and clumsiness was very marked. The left pupil was larger than the right; both pupils reacted to light and in accommodation, and there was no paralysis of the extra-ocular muscles. The eyegrounds showed choked disk with 3 and 4 dipters of swelling. Astereognosis was present in the right hand although the sensibility (all forms) was retained. The biceps and triceps reflexes were normal on the two sides; the patellar and Achilles reflexes were increased on the right.

Laboratory tests, including roentgen ray and the Wassermann reaction gave no indication of special features and a diagnosis was made of brain tumor of the left side, probably cortical.

Jan. 16, 1922, Dr. Frazier performed an osteoplastic flap exposure over the left rolandic area. On reflecting the calvarium the dura was found extremely tense and very vascular. To relieve intradural tension 15 per cent. salt solution was given intravenously and ventricular puncture was performed. There was immediate relief of tension. The dural flap was reflected and a tumor found growing from the dura and having the reddish granulomatous, vascular appearance of a sarcoma. It did not invade the cerebral substance. It was removed by blunt dissection, hemostasis being secured by silver clips. A section of the dura 6.5 by 8.5 cm. in area was removed with the tumor, and the defect filled with fascia lata graft. Some strips of celloidin were implanted for drainage. Transfusion was performed. The patient was conscious at the end of the operation.

Two days after the operation, the patient began to experience movements of extension of the wrist, about twenty times a minute. These continued for two days and then ceased. There was almost complete loss of power in the right arm, although at the end of five days, some movements were observed when the other arm was exercised. Two weeks after operation there was a fair return of power. The bone flap had ridden up for some unexplained reason and had to be wired down; after this recovery was uneventful.

On discharge, Feb. 11, 1922, it was noted that the patient had no headache, that vision had improved and the swelling of the nerve heads had partially subsided. He had more difficulty in speaking, and greater weakness in the right arm and leg than on admission. At the present time he has regained much of the buoyancy of his walk, his grip is strong, he recognizes objects well, and has no difficulty in talking or writing.

The tumor showed an abundance of cells with many small thin-walled vessels. The cells had large, deeply staining vesicular nuclei and were arranged in whorls. The histologic diagnosis was endothelioma.

GLIOMA OF MEDULLA; REPORT OF CASE WITH EXHIBITION OF SPECIMEN. DR. WALTER FREEMAN.

The patient, a man aged 41 years, complained of weakness, vomiting and numbness. In November, 1921, he had spells of involuntary movements of the feet. Later he noticed that he fell asleep very readily and was difficult to rouse. His head ached and he felt dizzy. In January, 1922, he began to stagger to the right and seemed to be falling, although he never really fell; yet he could not keep on the sidewalk. In this month he had a severe attack of hiccough that lasted four or five days. Numbness began in his hands and feet on both sides, and he had paresthesias over the back of the head and behind the ears. He had the sensation of "walking on mud." At this time he began to speak thickly and sometimes made mistakes, reversing words or transposing them, although they were all put in and the context would clarify the meaning. He did not omit words or syllables, use the wrong word, skip words, or insert meaningless words in the sentence. For instance, he might say "iron hard" for "hard iron." He was unconscious of these slips at first and blamed his secretary for mistakes she had not committed. He had no aphasia. In reading he would sometimes read parts of paragraphs repeatedly.

In March projectile vomiting began and his eyesight became dim. A curtain seemed to fall before his eyes and things seemed to revolve without disappearing. About March 20, he began to have difficulty in swallowing with regurgitation of liquids through his nose, and saliva collected in his mouth. Shortly before admission, April 14, 1922, he began to bite his tongue. He felt as though he were turning to the right.

Physical examination revealed a well developed adult male of high intelligence. Speech was noticeably thick and he was continuously expectorating saliva. The blood pressure was: systolic, 95; diastolic, 65. There was no choking of the optic nerves; slow nystagmus to the right was present, less to the left. Hearing was normal. The palate appeared to act normally, but the tongue deviated slightly to the right; phonation was irregular. Vomiting and hiccoughing recurred at intervals. The sternomastoids appeared strong but both trapezius muscles seemed weak. There was some weakness of the left extremities with increased reflexes, ankle clonus and a Babinski sign. Sensibility in the limbs, including the sense of position, was normal, but behind the right ear and over the back of the head there was distinct hypesthesia. Spinal fluid was under normal pressure. No spinal subarachnoid block.

Operation was performed by Dr. Frazier, April 21, 1922. On account of the serious condition of the patient, local anesthesia was employed. The usual crossbow incision with suboccipital craniectomy was made. The field was dry, and a very satisfactory exposure was made. It was noted that the right side of the medulla bulged, and this seemed to be due to an intramedullary growth. The operation was concluded with the patient in excellent condition. The systolic blood pressure had risen from 95 to 145 during the course of the operation, and the tongue could now be protruded in the midline.

After the operation, however, the patient was absolutely unable to swallow, had to be fed by stomach tube, and five days later died, apparently from vascular collapse.

The specimen shows an infiltrating growth of the right side of the medulla and upper spinal cord, involving to some extent also the left side. Histologic examination by Dr. Spiller confirmed the diagnosis of glioma.

TWO CASES OF PERIPHERAL NERVE TUMOR. DR. F. C. GRANT.

CASE 1.—A man, aged 39, for the past year has noticed a rather rapidly growing lump on the outer side of the right arm just above the elbow. This swelling appeared rather suddenly and he was told that he had a rupture of his muscles. No history of injury or hypodermic injection could be obtained. There was no anesthesia in the forearm or hand, no paralysis and very little weakness in the extension of the wrist and fingers.

A tumor of the musculospiral nerve was diagnosed. At the operation Dr. Raydin discovered a tumor, 3 by 3 by 8 cm. in size, apparently arising from the sheath of the musculospiral nerve. The brachial radialis muscle was spread out over the tumor. The nerve was dissected free from the tumor without damaging it. The tumor was definitely encapsulated. After removal of the tumor, wrist drop developed with increased weakness of fingers; these disappeared within five weeks after the operation. Dr. Walter H. Freeman of the Philadelphia General Hospital diagnosed the tumor pathologically as a perineural sarcoma.

CASE 2.—A woman, aged 46, complained of a lump on the inside of her right arm. Nine years ago she had an injection of typhoid vaccine at this site and for the past three years she has had a small lump in that region. This has gradually increased in size; it is painful to touch and causes pain when her arm is used excessively.

On examination a nodule was found on the inner aspect at about the middle of the right arm. Tingling in the thumb and first finger ensued when it was pressed upon. There was weakness in the same region, but no definite paralysis or anesthesia.

At the operation, an incision was made along the anterior aspect of the arm; the mass was explored and thought to be a neuroma involving, but not severing, the median nerve which passed along the deeper aspect of the growth as a flat band. This was excised with a section of nerve at each end; the severed ends of the nerve were brought together by dissecting down and up, and by flexing the arm at the elbow.

The tumor was circular in shape and about 3 cm. in diameter, well encapsulated and rather firm. It apparently sprang from the nerve sheath and displaced the nerve fibers laterally. The histologic diagnosis was endothelial sarcoma of the median nerve.

A week after operation she presented the typical picture of a median nerve paralysis in the right hand. Complete return of function in the median nerve occurred eight months after operation.

DISCUSSION

DR. W. W. KEEN: There must have been such a wide gap between the two ends of the nerve that I would like to ask how the nerve was anastomosed with such an admirable result in restoration of function.

DR. CHARLES H. FRAZIER: We resort to two methods to compensate for defects, nerve stretching and posture. By continuous stretching over a period of fifteen to twenty minutes we can compensate for 2 or 3 cm. and by flexion of the forearm on the arm, we can compensate for as much more. There are, therefore, really no technical difficulties in overcoming a defect of 4 or 5 cm. Apart from the technical matters, the case interested me as an example of a nerve lesion incidental to the use of hypodermic medication. There have been a number of instances of nerve lesion following introduction of salvarsan. Dr. Dean Lewis of Chicago reported two or three cases not long ago. This feature of the case interests me most, as it is recorded that the woman had received an injection of typhoid vaccine directly over the site of the median nerve; she remembered that shortly afterward she developed weakness in the flexor muscles of the forearm and hand.

VENTRICULOGRAPHY FROM AN EXPERIENCE WITH THIRTY CASES. DR. F. C. GRANT.

This will be published in full in a later issue of the ARCHIVES.

Book Reviews

NERVOUS ILLS, THEIR CAUSE AND CURE. By BORIS SIDIS, M.A., Ph.D., M.D. Cloth. Price \$3 Net. Pp. 379. Boston: Richard G. Badger, 1922.

This book has not a few merits, not the least of which is its criticism of Freud and the Freudians. The author says that psychoanalysis, with which term Freud and his adherents have baptised their sexual theories and metaphysical wish-speculations, is savage and barbaric. Dr. Sidis' own thesis is much simpler. For him the impulse of self-preservation is at the basis of all animal life. He well says (p. 19) that from the simplest lump of protoplasm, constituting a microbe, to the highest form of life, such as man, one meets with the same primitive life tendency—the impulse of self-preservation. Throughout all animal creation one important purpose runs, and that is the preservation of life. This impulse has two aspects, a positive and negative. In the positive form the impulse is to keep the individual alive; in the negative, to preserve the individual from injury and death. This negative impulse expresses itself in the form of *fear*. It is the morbid activity of this instinct of fear that lies at the root of all the nervous ills. This, in brief, is the thesis that is expounded by Dr. Sidis.

In some, if not all, of this we are at one with Dr. Sidis. The emphasis put on sex by Freud and his imitators is mischievous and misleading. There are other instincts than the instinct of sex. The Great War clearly proved (what, indeed, needed no proof for any observing man) that the instinct of self-preservation, with its attending emotions of fear, cowardice, self-respect and self-reproach, is far more potent than the instinct of sex to cause the gravest psychoneuroses. But since the war made this truth so manifest it would seem that the time had come for these writers to keep their minds of sexual subjects, for a while at least. Dr. Sidis' book will help them to do this, and we believe that its influence in this respect will be altogether for good.

Dr. Sidis holds that fear is not a matter of belief. It is one of the most fundamental of animal instincts, having its roots deep down in the organism. Throughout his book he traces the action for good and evil of this fear instinct, and he does it, in the main, in an interesting and illuminating way. It is this that gives his book a truly philosophic tone.

The inferences which he draws from this idea in the domain of mental pathology are always engaging and often convincing. For Dr. Sidis this instinct of fear—this disturbance or perversion of the instinct of self-preservation—is the root of all evil. Whether the reader always agrees with him or not, he will find much that is novel and suggestive in the author's treatment of this subject. For our part we agree with him very often, and do not hesitate to recommend his book to neurologists.

We wish we could say that the book had no faults, but in this respect it is a very human document. Although he disclaims being a psychoanalyst, Dr. Sidis has some of the faults of that cult. The chief of these is his use sometimes of a very obscure and cumbersome phraseology. He evidently belongs to those who think they can venture at will into the dark realms of the subconscious. This does something to spoil his writing for us. When we met with the sen-

tence. "abnormal suggestibility is a disaggregation of consciousness," we were tempted to throw down the book in despair; and when a few pages later we read something about "reintegration in consciousness," we were not much encouraged to continue. Such words have no meaning for us, and we wish that the author had seen fit to clothe his ideas—which indeed are very plain and simple—in a plain and simple style.

But Dr. Sidis is not always obscure. He describes, with a truly dramatic touch, the supreme selfishness of psychopaths. The paranoid impulse in them is expressive of the disturbed instinct of self-preservation and fear. He thus denies an "inferiority complex," and says that the patient's real mental state is fear that his sense of superiority will be humbled. But is not this paranoid mood a sort of "defense reaction," as expressed in the lingo of the present day?

Much importance is attached to what the author calls the "hypnoidal" state, which is apparently the stage 'twixt sleep and wake, and is the same that some older writers have called the "predormition." At least, so it appears to us, although Dr. Sidis may not acknowledge the identity, for he claims the merit of having originally described and utilized this state. It is not a state of true hypnosis, although one apparently in which suggestion is easily used.

We do not quite agree with Dr. Sidis in his rather caustic criticism of the doctrines of heredity, but we think he is probably right in his poor opinion of eugenics. It is indeed refreshing to have some one speak up plainly and tell us what he thinks about the craze for legalized eugenics, which is only another name for universal maternalism.

We think that Dr. Sidis' book is too long. It contains too many tedious case reports, and is full of repetitions. It could easily have been compressed into one half the space, and much improved by the process. In saying this we are speaking for a neurologic world which is harassed by a multiplicity of writings. But it is a book worth reading.

THE KINGDOM OF EVILS; PSYCHIATRIC SOCIAL WORK PRESENTED IN ONE HUNDRED CASE HISTORIES TOGETHER WITH A CLASSIFICATION OF SOCIAL DIVISIONS OF EVIL. E. E. SOUTHARD, M.D., and MARY C. JARRETT, with an Introduction by Richard C. Cabot, M.D.; a Note on Legal Entanglement as a Division of Evil, by Roscoe Pound; and an Appendix on Legislation in Relation to Mental Disease, by Frankwood E. Williams, M.D. Cloth. Price, \$5.50. Pp. 708. New York: The Macmillan Company, 1922.

This book with its long and characteristic title was not quite completed at the time of Dr. Southard's death. It is evident that the affection of his friends gathers round his memory.

Book I gives full case records in an attempt to describe the public, social, and individual spheres of social work. Book II has as a special feature a division of "The Kingdom of Evil" into five states, Disease, Ignorance, Vice, Legal Entanglement, Poverty. The last five of thirty-eight illustrative records are examples of so-called pure types of each kind of evil, though the comment shows that the authors recognize that types are never entirely pure. The example of a problem in "Ignorance" follows: John Henry, aged 15, was rescued from a dark cellar where his mother had kept him for five months. His mother previously had kept him back at school and persuaded the teachers that he was feeble-minded. The poverty-stricken family had moved eleven times in five years, and he had been kept from contact with other children. All this did not prevent him from receiving a mental rating of 16 years on the Yerkes-

Bridges point scale and of 15 years, 1 month on the Stanford-Binet scale. The records given in Book III illustrate eleven major forms of mental disease. Before leaving the subject of this "quarry" of case record material, attention should be called to three unrevised records of the Psychopathic Hospital social service in the appendix; the first is eleven pages long, the second forty-nine pages, and the third thirty-eight pages.

In Book IV there is a change from detailed description to the laying down of principles, and to comment on social work in general. Dr. Southard regards the chronic insane as "a comparatively small group of unfortunates," though they are the main interest of state hospitals and state laws. His interest spreads to the psychopaths, the eccentrics, those very slightly subnormal, and to the supernormal. "One out of every two cases of social trouble is a psychiatric case." And so "psychiatric social work can establish and is now establishing new principles for social work in general."

In the discussion of the problem of Evil, Dr. Southard's interest in and dependence upon Professor Royce is in evidence, and gives the clue to the choosing of the quotations from Job with which the book is sprinkled. His passion for playing with definitions brings a many faceted dealing with the Grail hunters and the dragon killers, the rebuilders and the destroyers in our public life, the alienist as distinguished from the psychiatrist, the sociologist from the physician, and the contrasting aims of nurse, social worker, and psychiatric social worker. The reader is told particularly how the technic of psychiatric case work could be applied to war, bolshevism, and industry. There is both interest and incompleteness in this book. It shows psychiatry in its present state of transition.

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SOME PROBLEMS IN EPILEPSY *

PRESIDENTIAL ADDRESS

WALTER TIMME, M.D.

Our third annual meeting brings us a step further along on the road we have chosen to travel, and as becomes seasoned travellers, we find that we need to discard many of the impediments which at first were thought necessary to our journey but which have since been proved to have merely bulk out of proportion to utility. As our society is pre-eminently one for research and not for the mere compilation of extant literature, even though this latter must of necessity be gathered in our bibliography, our programs must gradually be freed from all matter not truly savoring of original thought and work. The outlook seems, therefore, to be for less extensive but more intensive presentations; and so this year's offerings will be found to have, I believe, few of the characteristics of the ordinary symposiums on epilepsy. We do not feel that the entire subject must be covered in all of its aspects, but only in those on which more light or a greater approximation than heretofore to apparent truth may be had.

The very first difficulty—and there were many encountered—that confronted your program committee, was the choice of a comprehensive title for the subject before the society. The term epilepsy as it was commonly understood was immediately found to be inadequate, for while the convulsive seizure was uppermost in the mind as the concomitant of epilepsy and was the factor largely to be discussed at this year's meeting, yet it is trite to say that, in the opinion of many, some epilepsies may have no convulsions at all in the ordinary acceptance of the term. On the other hand, many convulsive seizures that seem superficially to simulate an epilepsy, such as those occurring in uremia, diabetes, tetany, and many of the other toxæmias, may possibly not be truly epileptic. And so both terms, epilepsy and convulsion, had to be introduced into our title. It is not my function to differentiate or to define, but merely to point out the problem. You will find on our program, therefore, discussions on uremic and other toxic convulsive states and possibly even an attempt at a differential diagnosis between them and the epileptic. We shall also have exceed-

*Read before the Association for Research in Nervous and Mental Diseases, December 28, 1922.

ingly important, not to say interesting, reports of the production of convulsive attacks in animals simulating the epileptic attack, by various surgical and pharmacodynamic means. Immediately occurs the thought—if surgical experimentation and pharmacodynamic assault release a mechanism in either case producing substantially the same manifestations, the approach to such a corticomotor release may possibly be along many other paths as well. Investigators are more or less in agreement that such motor release may be occasioned by various toxins, both endogenous and exogenous—witness the eclamptic and uremic seizures, wormwood and strychnin poisoning; or by infection—syphilitic, encephalitic; or through metallic poisons, lead and mercury—or through a variety of other causes—all of which are to be separated from the anatomic causes produced by various forms and sequelae of traumatic insult, tumor, and other local irritative lesions in great number. And then, apart from these two great groups of causes of the convulsive state there is added the group of so-called idiopathic epilepsy or genuine epilepsy, with a convulsive manifestation which you will hear, during these two days, compared to the convulsive seizure of the two great pathologic groups mentioned. Are the mechanisms released in the three types the same, or do they differ in essential particulars as well as in their pathogenesis? To add to the complexity, some of us believe that instead of, or indeed in addition to, the motor release in genuine epilepsy there may be a pathologic release in other domains and on other levels than the purely motor cortical. So that in the receptors of the special senses, and other “gnostic” centers, manifestations occur with attendant specific characteristics such as peculiarities of taste or smell or vision or hearing; or again with behavioristic and moral, intellectual and psychic and symbolic disturbances, seen as the so-called epileptic equivalents—a term which degrades them to a subsidiary position, and is only just as true as though we called the “fit” itself the motor equivalent of a disturbance on the higher level. Descending to the lower levels, are there not also disturbances in the basal ganglions, in the extended vagus system and even in the various segments of the cord which lead to more or less circumscribed motor and sensory symptoms that are due to a release of some mechanism perhaps similar in its origin, but different in its point of application? The motor cortical release which, by reason of its dramatic intrusion, startles our senses into its acceptance as the “whole thing” has, by virtue of this self-advertisement, for ages dominated our conception of genuine epilepsy. Is it for this reason that the comparatively smaller and less insistent gestures have escaped their true valuation? That is to say, is a muscular fibrillation, or a myoclonus, or a subjective segmental transitory pain without any demonstrable lesion, one of the forms of release produced

in genuine epilepsy when the point of low threshold is a part of the spinal cord? It is possible that some of our theses will cover this point. As for the vegetative level, now that biochemistry is beginning to assume such magnificent proportions in our medical uncomfortableness, that is the line on which—having failed in all others—we are now concentrating our attacks. If the reason for a genuine epileptic seizure—no matter on what point it is centered—is one of lowered threshold of the synaptic junction or other salient point of defense of the nervous system, then is such lowered threshold determined by a critical change in the biochemistry of the circulating fluids, or else of the neural elements? Many observations of changes in the coagulation time of the blood, in the alkiline reserve, in the organic and inorganic retained salts and split protein products, have come down to us as some of the underlying base-stones of the lowered threshold theory. As a corollary, what abnormal function of the glands of internal secretion could account possibly for such biochemical changes? One must have great optimism to face courageously these problems and to arrive at a comprehensive view of what we have termed genuine epilepsy. Your program to-day has on its rolls some such intrepid souls, and we cannot sufficiently convey to them our appreciation of their contribution to our science.

As my function is largely today an inquisitive one, as you may have noticed, may I add to some of the simple questions thus far propounded, this: If the genuine epileptic attack—no matter of what nature and focus—is due to a lowered threshold on the part of the neural elements or their structure, is such low threshold coefficient inherent in some constitutions apart from any biochemical, toxic, or other factor; so that certain individuals will respond with an attack psychic, intellectual, behavioristic, convulsive, vagal, or vegetative, or a combination of these more readily than others? If this is so, then are such individuals even without such attack (as we have been told by some observers that they are) different in their adaptability to their environment and to their problems? Is there an epileptic personality *per se*, with not only no necessity, through an attack, for diagnosis, but actually without attack? Perhaps this part of our problem will be solved for us tomorrow.

From these more or less lucid questions and expressions, one thing, I believe, stands out prominently and that is, that to call a patient "epileptic" means almost nothing in an explanatory sense—it is merely calling him a name. If my questions were all answered affirmatively we were all potential epileptics; and yet I doubt not, many of us would answer affirmatively and consider epilepsy an expression of a failure to overcome one of the difficulties that the race, in reaching for higher

and more specialized development, encounters in its upward gradient. It would, in such a sense, be a hall-mark of the non-adaptability of the unfit in this drive for the ultimate.

I have already encroached too long a time on your patience and on our exercises, but something still remains to be said. If a tendency to a wider, more extensive application of the term epilepsy is apparent, it becomes increasingly our duty either to take the fear and sting from the term epilepsy—for its mere individual application is fraught with as acute perils as was the mediaeval term “heresy”; or else to invent a new name to cover the situation.

Apologetically, and in fear and trembling, may I venture to suggest that while the term epilepsy be retained to apply to all the symptoms complex that are at present thought to be due to what the term epilepsy connotes, namely, “a seizure upon” the individual through extraneous causes, such as irritative lesions and drugs and toxins, yet that a new term connoting a seizure due to causes *within*, and of the biological unit *per se*, be used to cover the inadequacies and deficiencies of the mechanism of the biological unit in its selfdefense. Such a term might be “endolepsy.” Following the adoption of the complementary, and to an extent the antithetic, terms “epilepsy” and “endolepsy,” we would have at once a separation on a more or less rational basis, of the many conditions heretofore known by the many hyphenated expressions that fill our discussions of the subject, into two great classes. It will tend to clarification, to definiteness of picture, to less diffuseness of argument, and with a distinct end-product for our experiment and ratiocination to prove or disprove.

Gentlemen, our greetings to you on this third anniversary meeting of our association, and our thanks for the great interest and support and assistance that you have given since the inception of our organization. Your New York confreres extend you their greetings and bid you welcome.

THE ECOLOGY OF EPILEPSY

I. EPILEPSY IN COLONIES *

CHARLES L. DANA, M.D.

NEW YORK

Epilepsy is not only a medical problem, it is also a social and even a zoological one; and it appeals emphatically to preventive medicine, as all incurable diseases do, or should do. These points of view are included under the term Ecology, a science that deals with groups, and with the relation of these groups to the environment. It calls for a broader view than do the ordinary studies of the nature and cause of the disease.

For epilepsy has apparently a definite injurious influence on social life and economics as well as on the individuals who suffer from the disease. My aim has been to study the disease in bulk; with reference to the social effect of massed epilepsy on society, and of the units of the mass upon each other. Another line of attack is that of inquiry into the racial, geographical and social, and even evolutionary conditions that have led to the development of epilepsy in man and animals. For it seems that epilepsy has existed in every human race, and probably in all vertebrate animals, since the beginning of vertebrate and human life. It is along this last line that Dr. C. B. Davenport has made investigations.

The object of this paper is an investigation of epilepsy in mass; i. e., in epileptic colonies. The inquiries bore on the relations of the colonies to the community about them, the personality of colonies of epileptics; and the effects of segregation on the epileptics themselves; whether or not the patients with epilepsy thrive en masse.

In response to a questionnaire letter to the medical directors of the nine epileptic colonies in the United States, I received lengthy and carefully thought out replies from: (1) Dr. A. S. Priddy, State Colony of Virginia; (2) Dr. A. B. Bass, State Colony of Texas; (3) Dr. F. O. Butler, of Sonoma State Home of California; (4) Dr. Benjamin W. Baker, of the School for Feeble-Minded, of New Hampshire; (5) Dr. D. F. Weeks, of Village for Epileptics of New Jersey; (6) Dr. William T. Shanahan, of Craig Colony, New York.

The opinions were unanimous that the segregation of epileptics in colonies was not harmful, but economically satisfactory to the surrounding communities, and that epileptics when living together did not react badly upon each other; but were on the whole better and happier.

* Read at the third annual meeting of the Association for Research in Nervous and Mental Diseases, New York, Dec. 27, 1922.

The special advantages in opportunities for education, occupation and treatment have been demonstrated and are familiar to all. Incidentally, there might come up the question whether eventually an epileptic infection of the community occurs, i. e., whether in time there develops more epilepsy among the people living in and about an epileptic colony. It must be, that occasional irregular intercourse takes place between the patients and outsiders.

The establishment of epileptic colonies is proved to be a successful way of dealing with the disease, but in almost all cases the directors are dissatisfied with the inadequacy of state support. There are, I should say, nowhere in the United States ideal or model colonies for epileptics as there are for mental defectives and the insane, and nearly all the directors plead for more help, better equipment and fewer mental defectives, for the mental defective is grouped with the epileptics in many states.

This epilepsy mass has certain special features that give it a kind of personality of its own. Some of the characteristics of the group are these:

1. The group is made up of individuals who are on the whole not only incurable but steadily deteriorating. It is a dying forest growing in a fertile soil. In this respect, the group differs from that of the insane, and of the feeble-minded; the former has hope of cure, the latter of improvement.

2. There is a prevalent querulousness, irritability, dislike of discipline, and of the prolonged effort which leads to manual skill and to a definite technical occupation. The special character of the chronic deteriorating epileptic gives its quality to the atmosphere of the institution.

3. There is the greatest possible range of intelligence from idiocy to cleverness, and great variation in behavior from precise rectitude to delinquency.

4. The ever present element of the convulsive seizure gives a certain atmosphere, involving anxiety, expectancy and dramatic episodes; all toned down by familiarity with the situation.

These characteristics of massed epilepsy do not conflict with the fact that the seriously epileptic are better occupied, better educated and lead a more effective and happy life than if they were in a normal community. From epileptic colonies also are coming studies which will show us, we believe, how to prevent and lessen the prevalence of the disease.

The investigations of Professor Davenport and others show, as already stated, that the convulsive seizure is present in all levels of vertebrate life, and is thus a definite biologic factor in the evolutionary

process. If that is so, it seems probable that the convulsive seizure must have some meaning or purpose in the building up of structure and function. An organism may have to be keyed up to the point of explosion in order to be effective. The existence of epilepsy in groups and colonies may furnish better opportunities for making studies that will throw light upon this interesting phase in the study of the convulsive seizure.

If the epileptic represents a specific type anthropologically and zoologically, there may be found some indication of it in the somatic make-up, just as Dr. Lewis seems to have shown these somatic peculiarities in the paranoid and praecox types of mental disease. There should be a comparative study made of the whole body including all the organs and tissues of epileptics, in order to see if there is any common abnormality. This abnormality may not be in the nervous system, and it may be shown that epilepsy is not a disease of the nervous system but of the whole soma. This is more probable than that there is something peculiar and characteristic in the personality.

THE ECOLOGY OF EPILEPSY

II. RACIAL AND GEOGRAPHIC DISTRIBUTION OF EPILEPSY *

C. B. DAVENPORT, PH.D.

COLD SPRING HARBOR, LONG ISLAND, N. Y.

Idiopathic epilepsy may be defined for the purposes of this paper as a tendency, more or less periodic, to convulsions with loss of consciousness, without obvious exogenous factors, and in persons who have an ordinarily well developed brain. Our problem is: what is the distribution in the human species of this tendency to epilepsy?

Three general matters may be discussed at the outset. First, idiopathic epilepsy is not always easy to separate from epilepsy with exogenous factors; for there may be such factors all unknown to the student. Also, even statistics of army recruiting and missionary hospitals do not distinguish epilepsies due to different causes. We shall have to be content with what is available, unsatisfactory as it is. The defect is not so serious, however, since even in cases of epilepsy in which an irritating or inducing "cause" can be distinguished, we can not deny the probable existence of predisposing constitutional factors. Purely exogenous epilepsy is probably relatively uncommon.

Second, epilepsy is one of the most widespread of symptoms. It is found in all races of men; it is found in the animals that we know best—the domestic animals. Examples of these have been cited by L. P. Clark¹ (1904) from the works of Féré and Gelineau. The horse, cat, dog, ox, pig, occasionally the rabbit, and even birds like poultry and the canary may show epileptiform convulsions. Evidently the possibility of clonic contractions of the muscles depends on an architecture of the central nervous system that is common to all of the higher vertebrates; and upon a mechanism which reacts similarly to disturbance of that system. If this is true for mammals as a whole we may expect it to be true for all human races.

Hirsch² (1886) indeed says of the whole group of nervous diseases "there is no other so widely spread in time and space and no other has such pronounced, ubiquitous character as epilepsy. The disease is described in all the early medical writings, in the com-

* Read at the third annual meeting of the Association for Research in Nervous and Mental Diseases, New York, Dec. 27, 1922.

1. Clark, L. Pierce: A Comparative Study of Idiopathic Epilepsy in Animals and Man, New York M. J. **80**:1105 (Dec. 10) 1904.

2. Hirsch, August: Handbuch der historisch-geographischen Pathologie, Ed. 2, Stuttgart, 1886, Vol. III, p. 371.

pendia of the Arabian and of the western doctors of the middle ages; in the medical literature of the sixteenth to eighteenth centuries. . . . The medical-topographical reports of the present time bear witness to the universal incidence of epilepsy over the whole inhabited surface of the earth."

Third, though epilepsy is ubiquitous it does not follow that its proportional incidence is the same in all populations. There are several considerations why we should expect the incidence to vary.

1. Idiopathic epilepsy implies a constitutional basis. Studies by Dr. David F. Weeks³ and me have, we think, demonstrated the presence of an hereditary factor which acts as a Mendelian recessive. Recessive traits tend to become commoner in a population as it becomes more inbred, provided the defect was present in the ancestry. Thus one of the islands off the coast of Maine, which has been studied by the Eugenics Record Office, reveals an inbred population with a high incidence of epilepsy which can be traced back to the common ancestor of most of the people of the island. Many years ago Dr. Bemiss⁴ (1858) reported to the American Medical Association on a collection of 833 consanguineous marriages producing 3942 children, and of these 15 per mille were said to be epileptic. Such facts, which might be multiplied, suggest that we might expect some races in which consanguineous marriages are exceptionally common to show a high incidence of epilepsy.

2. Again, it is sometimes alleged that the drinking of much alcohol induces epileptic attacks and these may recur in the descendants of such imbibers. The epilepsy becomes idiopathic in this strain. If there is anything in this view, countries where little alcohol is used, e. g., India and Turkey, should show a low incidence of epilepsy; countries where strong alcoholic drinks are used to excess, as formerly in Scandinavia, should show a high incidence of epilepsy. At any rate, the existence of the hypothesis justifies the testing of it.

In view of these considerations it becomes desirable to bring together what is known about the rate of incidence of epilepsy in different countries and this is attempted in the following paragraphs from the literature. I take this opportunity to acknowledge the great assistance given by Miss Mabel L. Earle of the Eugenics Record Office in reviewing the literature of this subject.

3. Davenport, C. B., and Weeks, D. F.: A First Study of Inheritance in Epilepsy, *J. Nerv. & Ment. Dis.* **38**:641, 1911; *Bull. No. 4*, Eugenics Record Office, November, 1911.

4. Bemiss, S. M.: Report on Influences of Marriages of Consanguinity upon Offspring, *Trans. A. M. A.* **11**:321, 1858.

INCIDENCE OF EPILEPSY IN DIFFERENT COUNTRIES OR RACES

A. NEGROES

Hirsch² (1886) concludes that epilepsy appears in the same form and with equal frequency among the black races of the west coast of Africa as in Europe and other countries. Unfortunately our information as to conditions in Central Africa lacks detail.

Chassaniol⁵ (1865) found epilepsy to occur in Senegal much as in Europe, and Bonius (1882) writing from the same place states that epilepsy is of frequent occurrence. Clarke⁶ (1860) speaking of the Gold Coast says epilepsy was occasionally brought to his notice.

In the United States we have numerous "negroes," but they are largely mulattoes and, living in contact with the whites, have acquired their vices. Large numbers of such negroes were examined in the army draft of 1917 to 1918 by the same examiners as the white men, and the findings were studied by Love and Davenport,⁷ in 1920. It may be instructive to compare the incidence of epilepsy in the two races. In those areas of the Southern States which are inhabited by 45 per cent. or more of "negroes" there was found a lower rate for epilepsy (5.13) than for the prevailingly white Southern agricultural districts (5.65) or for the "mountain white" areas of the south (6.28). This result suggests that "negroes" are somewhat (but only a little) less affected by epilepsy than whites. This accords with a statement of Searcy, who was familiar with the negro race in the Southern States and is quoted by Spratling⁸ (1904). He says "it is a generally expressed opinion that insanity and epilepsy are rapidly increasing among negroes." This he ascribes to the removal of the beneficial physical effect of slavery; but it may be equally explained on the ground of increasing hybridization with whites. Modern statistics concerning epilepsy of negroes in the United States are vitiated by the inclusion of mulattoes in the group of negroes.

A careful inspection of Table 1 shows, first, that the rate for epilepsy in the U. S. Army was maximal in the army in the United States in 1918. The rate was high, probably because the army was maintaining in "development battalions" many epileptics who should not have been in the army. The rate was correspondingly low in the

5. Chassaniol: Contributions à la pathologie de la race nègre, Arch. de méd. navale. **3**:509, 1865.

6. Clarke, R.: Remarks on the Topography and Diseases of the Gold Coast, Trans. Epidem. Soc. of London **1**:114, 1860.

7. Love, A. G., and Davenport, C. B.: Defects Found in Drafted Men, War Dept., Washington, Government Printing Office, 1920.

8. Spratling, W. P.: Epilepsy and Its Treatment, Philadelphia, W. B. Saunders Company, 1904.

army in Europe, because these were picked men. In the United States in 1918, the colored rate of 7.23 per mille was a very high one and 2.3 times that of the whites.

During the World War the rate of admission to sick report for epilepsy varied with race as follows:

TABLE 1.—ADMISSION TO SICK REPORT, UNITED STATES ARMY, EPILEPSY *

Group	—Ratios per 1,000—	
	White	Colored
Calendar Year 1917		
Total, United States, Europe, etc.....	2.70	5.23
United States only.....	2.88	6.54
United States, only, regulars.....	3.04	5.49
Europe only	1.22	4.15
Native troops, Porto Ricans.....		0.52
White troops in Philippines.....	0.64	
Philippine scouts		0.54
Calendar Year 1918		
Total United States Army, military men.....	2.09	4.92
United States Army, in United States.....	3.12	7.23
United States Army, in Europe.....	0.81	1.49
White troops in Philippines.....	1.05	
Philippine Islanders		0.33
Hawaiians		0.86
Porto Ricans		5.00
Calendar Year 1919		
Total, United States Army.....	0.88	0.92
United States Army in United States.....	1.38	1.18
United States Army in Europe.....	0.67	0.92
White troops in Philippines.....	1.55	
Philippine scouts		0.12

* From Reports of the Surgeon-General, United States Army.

For the year 1917, when we were inducting hundreds of thousands of men into army service, the rate for colored troops in the United States (6.54) was again 2.3 times that for whites (2.88). For the year 1919, when the development battalions had been discharged, the admission of colored troops in the United States (1.18) was less than that of white troops (1.38). Apparently the discharges for disability on account of epilepsy, which in 1918 were (in the United States) 2.4 times as great for colored as for whites, had helped put men of the two "races" on the same plane.

To sum up, the apparent discrepancy between the record of drafted men and that of the army may be harmonized by the hypothesis that in areas with a large proportion of negroes the epilepsy rate was fairly low because the proportion of full blooded negroes was high; so, on the contrary, the "negroes" from other parts of the country, who are really for the most part mulattoes, show a great predominance of epilepsy over the whites. If this hypothesis were confirmed it would support strongly the theory that human hybrids in general are disharmonious, and show a preponderance of nervous defects over the

"purer" races. According to this theory, miscegenation increases the number of new centers of epilepsy; while inbreeding tends to increase the proportion of epilepsies in the descendants of one of them.

Considerable epilepsy was found by Pruner,⁹ 1847, in Egypt and from a table of Bertherand¹⁰ (1855) it would appear that among the Mussulmans only (who are forbidden to use alcoholic liquors) epilepsy is an important cause of death.

B. AMERINDIANS

Dr. A. Hrdlicka¹¹ made first hand physiologic and medical observations on thousands of pure blooded Indians of our Southwest. Of the Otomi (Hidalgo) Indians, who drink to excess, he says: "epilepsy and other neuropathic conditions are met with frequently." In comparing the Southwestern Indians with average white Americans he says: "Nervous diseases are relatively rare, except epilepsy." Dr. Hrdlicka sent a letter of inquiry to the physicians in the Indian service concerning the incidence of various diseases, including "epilepsy." Reports were received from 102 localities. Epileptics were reported from 47 localities, comprising a population of 70,723 Indians (including half-breeds). There were reported 146 cases, probably all grand mal, in these 47 localities; making a rate of 2.1 per mille. No cases were reported from the other 55 localities. If they had the same population as the 47 reporting epilepsy, the epileptic rate would be reduced to about 1 per mille, which is low. It seems probable that the reporting was far from satisfactory. In certain stations a fairly high rate was reported. Thus among 5,000 Sioux of the Rosebud Agency, twenty-three cases, or 4.6 per mille; among 6,690 Sioux at Pine Ridge Agency, South Dakota, seventeen cases, or 2.5 per mille. It is probable that in migratory tribes the epileptic children die off early. Among 6,600 Pima Indians, at Pima Agency, Arizona, one case is reported, or 0.15 per mille.

In the army draft there were found in South Dakota Indian reservations five cases of epilepsy or 4.3 per mille; in Arizona, Indian Section, (35 per cent. Indians) three cases, or 1.3 per mille; in the New Mexico Indian section (29 per cent. Indians) ten cases, or 8.1 per mille. In the entire "Indian" group, 4.3 per mille epileptics were found, which is less than the average for the whole country (5.2 per mille).

From available data it is probable that the epilepsy rate for Amerindians is slightly less than that for white persons in the United

9. Pruner, F.: *Die Krankheiten des Orients*, Erlangen, 1847.

10. Bertherand, E. L.: *Médecine et hygiène des Arabes*, Paris, Baillière, 1855.

11. Hrdlicka, A.: *Physiological and Medical Observations Among the Indians*, Washington, Smithsonian Inst. Bull. No. 34, 1908.

States. Whether there is a difference in this respect between full blooded and halfbreeds cannot be stated.

Hirsch (1886) states that epilepsy is reported to occur with special frequency in Brazil: but it is not stated that this is true for the Indian population. Also, no ratios are given.

C. MONGOLIANS

China. For this country we have only certain hospital statistics. Thus, in the Medical Reports, Customs Gazette of Shanghai,¹² years 1871 to 1877, are given data furnished by certain medical missionaries. Taking reports where at least one case of epilepsy was recorded during the year (showing that the diagnostic term constituted a class included in the report) we have 21,874 patients and of them sixty-six with epilepsy, or 3 per mille. In Report 58 (p. 74) one case of epilepsy is reported in 400 patients or 2.5 per mille. Dr. W. H. Park reports in 25,000 native patients at Soochow Hospital seventy cases of epilepsy, or 2.7 per mille. In 350 consecutive private cases were two cases of epilepsy, or over 5 per mille. Jeffreys and Maxwell (1910) estimate that epilepsy is probably no less frequent in China than in western lands. Hofmann (1913) finds among 312 insane patients of the J. G. Kerr Hospital at Canton, three or about 10 per mille were cases of epilepsy. Schofield (1882) reports that of 1,851 cases treated at Shansi, North China, eight were cases of epilepsy, or 4.3 per mille. At the Harvard Medical School¹³ in China, of 1,438 cases treated medically in 1915 to 1916, five were epilepsy or 3.5 per mille. These are close to the European proportions. Dr. Andrew H. Woods, of Peking Union Medical College, states that among 16,000 patients in the Hospital and outpatient department, he found fourteen cases of epilepsy or not quite 1 per mille. This fourteen does not include organic cases due to syphilis, tumor, embolism and trauma. Dr. Wood thinks that

12. Medical Reports, Customs Gazette, Shanghai, China, Dr. David Mansoris. Reports on the Health of Tahow and Tauwen-foo, No. 3, p. 35; No. 4, p. 24; No. 5, p. 26; No. 8, p. 13; No. 11, p. 25. Dr. A. G. Reid's Reports on Health of Handow: No. 4, p. 72; No. 5, p. 29; No. 8, p. 41. Dr. George Shearer's Report on Cases Treated in Private Practice and in the Kiu Kiang Dispensary for Chinese, 1871, p. 62. Dr. Jardin's Report on the Health of Kiu Kiang, 1875, p. 1. Dr. Scott on Health of Swatow, 1871, No. 11, p. 83. Dr. E. P. McFarlane on the Health of Ichang, 1880, No. 20. Report No. 58. Szemao, 1899; Report No. 59. W. H. Park's Report on the Health of Soochow, 1900, No. 30, p. 5. Dr. H. M. Allen, Health of Seoul, Korea, 1885, p. 17. Schofield, H.: Report of the Medical Mission at T'ai-Yuen-Fu, Shansi, North China, Shanghai, 1882.

13. Reports of the Hospital Department, Harvard Medical School of China, Methodist Publishing House. Annual Report No. 2, 1915-1916; Annual Report No. 3, 1915-1916; Annual Report No. 4, 1916-1917.

the incidence is really much higher, as few cases of petit mal are recorded, and the Chinese would not be likely to bring any but serious cases to the hospital.

Korea. Dr. H. N. Allen¹² (1885) reported on the health of Seoul: "Epilepsy is surprisingly common; the worst case yet seen, however, had seizures only twice daily. The disease is considered no bar to matrimony, which may account for its prevalence." In the first 6 months at Allen's Hospital, 7,234 outpatients were treated, 184 for epilepsy or 23.8 per mille.

Japan. In the Navy Department of Japan, "Surgical and Medical history of the naval war between Japan and Russia"¹⁴ (1904 to 1905) is a report on 53,657 cases of admission to sick report, eighteen being for epilepsy—this is 0.42 per mille. These were, however, presumably picked men; nevertheless, the rate is low as compared with the 2 per mille admission to sick report in the U. S. Army during the World War. The data establish the presence of epilepsy among the Japanese but do not enable us to judge of its rate in civil life.

D. EAST INDIANS

Caddy¹⁵ (1901) reports that in 1899, among European troops in India, out of a total average strength of 67,100 men there were eighty-six admissions to sick report for epilepsy, or 1.2 per mille; while among native troops, with a total average strength of 128,530 there were only sixty-three admissions, or 0.5 per mille. Macphail¹⁶ reports that of 7,534 patients treated in his dispensary and hospital in 1904, thirty-six were suffering from epilepsy or 4.8 per mille. In 1834 to 1838, it is stated by the Madras Medical Board Office¹⁷ that of 7,093 admissions of European troops stationed at Malabar and Canara, thirty-two were cases of epilepsy or 4.5 per mille; while of native troops, out of 11,282 admissions only eight were for epilepsy or 0.7 per mille. Of native troops stationed at Calicut: of 1,416 admissions, one was for epilepsy; and of the native troops at Mangalore, of 7,875 admissions six were for epilepsy; together 9,291 admissions, with seven epileptics, or 0.75 per mille. Again of native troops at Hurrayhur, 1832 to 1841, of 5,040 admissions, six were for epilepsy, or 1.2 per mille. Of 5,322 admissions of native troops at French Rocks, two were cases of

14. Navy Department, Japan. Bureau of Medical Affairs: The Surgical and Medical History of the Naval War Between Japan and Russia During 1904 to 1909. Tokyo, 1911.

15. Caddy, A.: Trans. Nat. Assn. for the Study of Epilepsy **1**:177, 1901.

16. Macphail, J. M.: Report from India, Trans. Nat. Assn. for the Study of Epilepsy **3**:212, 1904.

17. Report on the Medical Topography and Statistics of the Province of Malabar and Canara, Records of the Medical Board Office, Madras, 1844.

epilepsy or 0.4 per mille. Again, in the Mysore Division: of European troops, among 13,500 admissions, thirty-one cases of epilepsy appeared, or 2.3 per mille; of native troops among 27,085 admissions, eighteen cases of epilepsy or 0.7 per mille.

Bryden's statistics for the Bengal Presidency¹⁸ (1860 to 1863) show: for the European army (strength 43,000), out of 83,406 admissions 147 or 1.7 per mille were for epilepsy; for the native army (strength 37,800), the average admissions were 50,500 of which twenty were for epilepsy, or 0.4 per mille. Even in the jail population the native epileptic rate is barely half that of the European.

From all of these considerations it seems justifiable to conclude that epilepsy is less than half as common in India as in England or the United States; and India uses little alcohol as a beverage.

E. PHILIPPINE ISLANDS

The admission rate to sick report among native troops of the U. S. Army for epilepsy is about 0.33, or one-third of the rate for white troops in that country.¹⁹ In fact the admission rate for Philippine scouts is extraordinarily low. But this may be due to exceptionally rigid selection.

F. POLYNESIA

From Mahatly's report²⁰ we learn that two Fijians and two Solomon Islanders were admitted to, and treated for epilepsy at, the Colonial Hospital, Sura, Fiji. The Senior Medical Officer at Sura states that he has treated cases of epilepsy among Fijians while he was District Medical Officer on the Island of Taviumi and in the province of Ba, but the cases were never numerous and symptoms varied in no way from those among epileptics of other races, particularly Europeans.

Of native Hawaiian troops in the U. S. Army,¹⁹ averaging 2,783, during 1918 and 1919 there was an average admission for epilepsy of 0.54 per mille, which resembles the extraordinarily low rate of the Philippine Islanders.

Thomson²¹ (1854) found that in New Zealand no Maoris (who are racially the same as the Hawaiians) had in 1854 been admitted into the hospital on account of epilepsy. He then took some trouble to

18. Bryden, J. L.: *Vital Statistics of Bengal Presidency*, Calcutta, Government Printing Office, 1871.

19. War Department, U. S. Army, *Reports of the Surgeon-General*, 1918, 1919 and 1920.

20. Mahatly, A.: *Report from Colony of Fiji*, *Trans. Nat. Assn. for the Study of Epilepsy* 4:290, 1906.

21. Thomson, A. S.: *On the Peculiarities of Figure, the Disfigurements and the Customs of the New Zealanders*, *Brit. & For. Med. Chirurg. Rev.* 14:461, 1854.

ascertain whether the disease had been seen among the Maoris and he concludes: "As I could never meet with a medical man, nor an European who had seen a native with epilepsy, nor a native who could describe the disease, I am inclined to think it does not exist among them, or if it does it must be very rare." Since all the Maoris were derived from a few ancestors (sixteen generations ago) it is quite thinkable that the genetic factors for the epileptic reaction to inciting agents were absent from their constitution.

G. EUROPE

For the European countries various estimates have been made:

Switzerland is of special interest because of the high proportion of consanguineous marriages in the valleys of that mountainous country. Wyrsh²² (1921) estimates that at Zürich, at least 3 per mille of the population are epileptic. In a rapid survey of the canton Unterwalden with 30,000 inhabitants, fifty-seven cases of epilepsy were found, a rate of 2 per mille; but this is a minimum. Ammann, using mortality statistics, army statistics of 1884 to 1909, and statistics of institutions and schools, concludes that the epileptic rate for Switzerland as a whole is 5 per mille—about the same as for the entire United States.

Italy, as a peninsula, has long experienced more than the average amount of inbreeding. According to Binswanger²³ (1899), Morselli found among 5,000 Italians (excluding convulsions in the first two years of life) six epileptics or 1.2 per mille—but this is not significant in absence of further details. Of 10,000 Italian military recruits, 11.53 per mille are said to have been rejected on account of epilepsy—a high rate.

Germany. According to Binswanger the rate of admission to sick report in the Prussian Army for the year 1889 to 1890, was 2.1 per mille; for 1895 to 1896, 2.9 per mille. The increase of the rate is ascribed to better means of detection. Among the school children of Mecklenburg, an epileptic rate of 1.15 per mille was found by Tigges,²⁴ 1883; but this count was incomplete. Estimates of smaller areas have given as much as 2 per mille.

France. According to Jolly,²⁵ French recruit statistics indicate an epileptic rate of 1.6 per mille.

22. Wyrsh, Jacob.: Zur Frage der geographischen Verbreitung und poliklinischen Behandlung der Epilepsie, Schweiz. med. Wchnschr. **2**:1182, 1921.

23. Binswanger, O.: Die Epilepsie, Wien, Hölder, 1899.

24. Tigges, P.: Die Zahlung der Epileptischen in Mecklenburg-Schwerin im Jahre 1882, Allg. Ztschr. f. Psychiat. **11**:543, 1883.

25. Jolly, F.: Arch. f. Psychiat. **13**:311, 1882.

Denmark. No adequate census has been made of the epileptics in this country. An estimate was made of 10,000 giving a rate of 4 per mille; but Søren Hansen²⁶ concludes that 3,000 is a safer estimate, giving a rate of 1.2 per mille.

II. GREAT BRITAIN

Fragments of the results of examination of army recruits during 1917 to 1919 have been published.²⁷ In one section of London the rejections for disabling epilepsy were 0.3 per mille for 1,100 British, and 1.4 per mille for 1,370 Jews (p. 39). In another locality (p. 48), of 2,894 rejections, sixty were for epilepsy, or 20 per mille. In still another locality (p. 113), 37 per mille were rejected for epilepsy. Of 10,000 recruits examined in Scotland, 6.4 per mille were found to have epilepsy of some grade, and 2.5 per mille of a grade requiring rejection.

Jews. Fishberg²⁸ (1911, p. 329) concludes that, despite a predisposition among the Jews to neuroses, epilepsy is less common among them than among non-Jews of the same locality. This is asserted to have been found in Paris, in Vienna, in Frankfort, in Moscow, and at Craig Colony in New York State. Dr. Fishberg states that among 3,000 applicants for relief in the United Hebrew Charities of New York, there is an average of about six epileptics per year, or 2.0 per mille.

I. UNITED STATES AND EUROPEAN NATIONS REPRESENTED IN IT

The statistics of "Defects Found in Drafted Men"⁷ tell something about the distribution of epilepsy in the United States. It appears from the map, p. 92, that the greatest incidence of this disease is in the population of New England, especially Vermont, where the rate was 12 per mille; but also in New Hampshire, rate 7 per mille; Connecticut, 6.9 per mille; Rhode Island, 6.7 per mille; and Maine, 6.5 per mille. Only in Massachusetts was the rate (5.9 per mille) decidedly lower, probably through segregation of many cases in the institutions of this socially highly developed state. Other high rates were found in Maryland, 8 per mille, and Louisiana, 6.9 per mille. These states, except Maryland, contain a large population of French origin. Aside from the maritime districts of the United States (rate 7.10 per mille)

26. Hansen, S.: On Antallet af Epileptikere i Danmark, Ugesk. f. Læger 4:373 and 419, 1897.

27. Galloway, J. H. W. Kaye, et al. 1920. Report upon the Physical Examination of Men of Military Age by National Service Boards from Nov. 1, 1917, to Oct. 31, 1918. Ministry of National Service, 1917 to 1919. London, Stationery Office, Vol. I, 1920.

28. Fishberg, M.: The Jews: a Study of Race and Environment, New York, Charles Scribner's Sons, 1911.

the northern agricultural sections with a predominance of native white stock has the highest ratio (6.7): the mountain white stock of the Southern Alleghanies has also a high rate (6.3). The Southern white agricultural communities have a fairly high rate (5.7) and also the southern section of mainly Scotch origin (5.2). On the other hand, the sections occupied by many Finns, Germans and Scandinavians have a relatively small rate for epilepsy (2.9 to 4.0 per mille).

H. H. Laughlin has recently collected statistics on the relative proportion of the different nationalities who are inmates of American institutions for defectives. These are still in manuscript; he has generously permitted me to abstract certain data. Thus, in nine states, custodial institutions for epileptics found in Massachusetts, Connecticut, New York, New Jersey, Michigan, Ohio, Illinois, Iowa and Texas—all but one in the north—including 6,807 epileptics, the largest proportion in relation to quota (based on population of the race in the United States) was found among peoples from the Turks of Asiatic Turkey, 167 per cent. of quota. Next, immigrants from Great Britain, 145 per cent. of quota; and next in order: Portugal, (133 per cent.), Russia, Poland and Finland, (117 per cent.), Rumania, (114 per cent.) and Ireland, (107 per cent.). Rates below their quota were furnished by immigrants from Greece (45 per cent.), from Austria-Hungary (67 per cent.), Germany (73 per cent.), Canada (75 per cent.) and Italy (83 per cent.). The extraordinarily low rate for Scandinavia (14 per cent. of the quota) is partly due to the fact that none of the states in the Scandinavian part of this country maintain separate state institutions for epileptics. But we have already seen from the Army Draft that the Scandinavian rate for epilepsy is rather low.

TABLE 2.—SUMMARY OF EPILEPTIC RATE IN RACES AND GROUPS

Class, Nationality or Race, How Obtained	Rate per 1,000
United States:	
2,500,000 drafted men.....	5.15
Rural recruits	5.36
Urban recruits	4.79
Boston recruits	6.13
New York City recruits.....	5.62
Chicago recruits	4.53
Vermont recruits	12.72
Maryland recruits	7.98
New York Recruits.....	6.07
New Jersey recruits.....	4.32
Kansas recruits	3.77
Utah recruits	3.48
South Dakota recruits.....	1.20
Southern agricultural—mainly native white.....	5.65
Southern agricultural—mainly negro.....	5.13
French Canadian sections.....	6.11
Scotch settlements	5.18
German and Scandinavian settlements.....	3.99
Scandinavian settlements	3.87
Finnish settlements	2.86
Army, admission sick report, 1918, white (2,204,709).....	2.09

TABLE 2.—SUMMARY OF EPILEPTIC RATE IN RACES AND GROUPS.—*Cont.*

Class, Nationality or Race, How Obtained	Rate per 1,000
United States:	
Army, admission sick report, 1918, colored (174,846).....	7.23
Army, admission sick report, 1917, white.....	2.70
Army, admission sick report, 1917, colored.....	6.54
Scotland, Army recruits, 1917 to 1919.....	6.40
Great Britain	0.3 to 37.0
British in institutions of United States, 145 per cent. of quota.....	
Denmark, various estimates.....	1.2 to 4.0
France, recruit statistics.....	1.6
Germany:	
Admission to sick report, Prussian army, 1889 to 1890.....	2.1
Admission to sick report, Prussian army, 1895 to 1896.....	2.9
School children of Mecklenburg.....	1.15
Other areas	2.0
Italy:	
Morselli's count of 5,000 Italians.....	1.2
10,000 military recruits, rejections for epilepsy.....	11.53
Switzerland:	
Zurich, estimated	3.0
Canton Unterwalden, minimum estimates.....	2.0
Ammann's estimate for whole country.....	5.0
Jews:	
Applicants for relief, New York City.....	2.0
Russians and Poles in institutions of United States, 117% of quota.....	
Maoris of New Zealand.....	0.0
Hawaiian Islanders, admission to sick report, United States Army.....	0.54
Philippine Islanders, admission to sick report, United States Army.....	0.33
India:	
Admission to sick report, Europeans (1889).....	1.2
Admission to sick report, Europeans (1834).....	4.5
Admission to sick report, native Indians (1889).....	0.5
Admission to sick report, native Indians (1834).....	0.7
Missionary dispensary hospital (Macphail), 1904, patients....	4.8
Native troops, Calcutta, admission.....	0.75
Native troops, Hurryhur, admission.....	1.2
Native troops, French Rocks, admission.....	0.4
Troops at Mysore, admission (European).....	2.3
Troops at Masore, admission (native).....	0.7
Bengal Presidency, Army admissions (European).....	1.7
Bengal Presidency, Army admissions (native).....	0.4
Japan, Navy, admission to sick report.....	0.4
Korea, Outpatients of missionary hospital.....	23.8
China:	
Shanghai hospital, patients.....	2.5
Soochow hospital	2.7
Soochow, private cases.....	5.0
Shansi, North China, hospital.....	4.3
Harvard Medical School in China.....	3.5
Amerindians:	
Hrdlicka's statistics on 70,000 Indians.....	2.1
5,000 Sioux, Rosebud Agency.....	4.6
6,690 Sioux, Pine Ridge Agency.....	2.5
6,600 Pimas	0.15
United States Draft, South Dakota Indian reservation.....	4.3
Arizona Indian section.....	1.3
New Mexico Indian section.....	8.1
Negroes:	
United States Draft, Negro sections, defect found.....	5.13
United States Army, admission to sick report, 1917.....	6.54
United States Army, admission to sick report, 1918.....	7.23
United States Army, admission to sick report, 1919.....	1.18

SUMMARY AND CONCLUSIONS

The tendency for muscles, singly or in groups to undergo tetanic contractions, more or less periodically without obvious exogenous causes, is wide-spread among the higher vertebrates. Consequently, its causative factor must be conceived broadly enough to include mammals and birds. Hence, while alcohol, diet, focal infection and trauma may be, and doubtless are, causative factors in particular cases, yet no one of them is the essential factor. The factor that is most nearly universally present is some constitutional peculiarity of the body that is inherited, so that the tendency for muscles to go "spontaneously" into tetanus recurs with especial frequency in certain families. An, if not *the*, hereditary factor behaves like a recessive. It is a priori probable that certain strains or races differ in the rate of incidence of epilepsy by virtue of either an inherited constitution, or a difference in the incidence of one or more exogenous factor, or by virtue of both of these causes.

Probable low incidence of epilepsy was found among the primitive Maoris, perhaps because the constitutional factor had not been brought in by the few immigrant ancestors. Some Amerindians appear to have a low rate by virtue of the early elimination of those showing symptoms. Otherwise, the rate is possibly slightly below that of the United States generally. Probable low rates are found among the East Indians, possibly due to low incidence of alcoholism; also among full-blooded negroes of the Southern United States. Soldiers of Philippine and Hawaiian Island stock have low rates. Apparently the same is true for Japanese naval men. In the United States territory populated largely by Finns, Germans and Scandinavians, the rate is low. Prohibition Kansas, and states occupied by the antialcoholic Mormons, have exceptionally low rates for epilepsy. In general, both favorable endogenous and exogenous factors are probably responsible for low rates.

High rates are found, apparently, in Korea, which as a long inhabited peninsula has probably an inbred population in which the incidence of the endogenous factor has become intensified. Italy, also a peninsula, seems to have a high rate among military recruits. The rate is high in some parts of Great Britain, an island. If these rates are reliable they indicate the danger of inbreeding in a population with the endogenous factors. In the United States high rates of incidence are found in sections where there are many French Canadians. Also Maryland has a high rate; whether or not this is causally connected with the strong pro-alcohol sentiment in the state, can naturally not be determined.

In general, the statistics suggest that alcohol may be an important exogenous factor which influences the epileptic rate; but doubtless the endogenous or constitutional factors are no less important.

EPILEPSY AND THE CONVULSIVE STATE *

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"As the departments of science become more and more complex, definition is recognized as a scholastical task which makes for sterility." An epigram by Sir Clifford Allbutt, which like all such obiter dicta can only be half true. To define explicitly may be futile or impossible; to describe, to classify and to set boundary marks can be done with difficulty, maybe, but always with advantage to clarity of ideas.

The effort to define may fail in ultimate truth, but will show us often the scope of our endeavor and the nature of our objective.

The idea of epilepsy which came to us from Hughlings Jackson is that it consists in the occasional sudden excessive violent discharge of some nerve center, sensory or motor; to which it would seem useful to add that the phenomena produced are independent of volition and are accompanied by interference with the stream of consciousness. Events of this general character—and not all epileptic phenomena can be covered even by this ample cloak—happen under such varied conditions, and in such a motley company of diseases that the notion is forced on us that the convulsive state and transient paroxysmal changes in consciousness cannot represent any single disease entity in themselves. They are but the expression of an innate or induced instability of nervous centers from diverse causes, operating, it may be, through a common constant connecting mechanism. Therefore, it may happen that the direction of search should be toward the nature of that mechanism which allows nervous explosion to occur—a quest which must range from cardiovascular ataxia to chronic intoxication and the protean appearances of anaphylactic shock.

It is vital to recognize, on the one hand, the essential unity of all attacks of loss or impairment of consciousness often accompanied by convulsions, and, on the other, the heterogeneous nature of conditions which may give rise to such attacks.

We cannot too vigorously insist on the frequently fleeting character of such epileptogenous conditions, nor combat overzealously the portentous, and miserably untrue prognosis saddled on such disorders by the public and many of our profession.

We are so familiar with epilepsy produced through renal disease and the poisonings of an uneasy pregnancy, that by granting such epilepsies special titles, we have obscured the fact that epileptic events in

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certain individuals follow hard on the heels of intoxications and are curable by the removal of the toxic material. Ignoring these facts, we arbitrarily classify cognate, often identical, phenomena in a category of disaster, whereby the diagnosis of epilepsy has become synonymous with a sentence to perdition. Epilepsy has been described by some of the most eminent writers on the subject as a condition characterized by recurrent convulsions and terminating in dementia. Surely this statement reveals but a fragment of one end of the spectrum of this disorder. True, the context usually attempts to adjust the ill balanced values set out by this description, but the context is apt to be difficult and the definition easy of quotation, the result being the unwisely restricted viewpoint so often taken of the condition and the often foolishly lugubrious outlook imposed on the subject and his friends. These remarks have been directed at those who would damn the future of the patient suffering from occasional convulsions or their isolated auras, but of late a group of writers has sought to blacken by quite improper emphasis, the moral and mental antecedents of the unhappy persons liable to this dyscrasia.

Thus it has been asserted that the individual destined later to recurring convulsions is shackled by a character ethically depraved; that he displays an egocentric contraction of the entire emotional life; that he is a pathologic liar, a distrustful pertinacious fellow given over to weak rages and black vapors. I imagine that such a portrait has been painted in a badly lighted studio rather than from life.

Every physician, of however slight experience, sees Calibans and Timons who never had a fit, and men of high moral and intellectual quality subject to the disorders we now discuss. However, premises of this character, or lack of character, are necessary to permit the psychologic interpretation of epilepsy submitted by this screedy school. A personality so misshapen and so warped must in self-defense commit occasionally psychologic suicide, must be allowed to dip itself in Lethe, to banish the horrid awareness of itself; in short, to have a fit in which it seeks the Nirvana of the womb, to search again periodically for its pristine purity and innocence by the resuscitation of impulsive fetal movements! Job cursed the day he left his mother's body, but it has been left to the minor poets of a less humorous age to make him investigate the possibility of resuming an umbilical attachment! My translation of this theory is perhaps too simple—but here is its original kernel (I have no means of knowing whether or not it has felt the displeasure of an irate compositor): "We are justified in considering the essential nucleus of the epileptic fit an infantile unconscious striving of displeasure-pleasure pursuit ending in the final goal of a return to infancy, attended by a loss of consciousness and a convulsion; that the convulsion is made up of and flows out of the general striving of the

fetal and infantile tissues as expressed through the lower spinal centres in inducing simple and crude combinations of impulsive movements; that a study of the degree of development of unconscious infantile strivings in the emotional instincts, the desire for an infantile state of omniscience (sic) are paralleled by the kind and character of impulsive movements found in this infantile period of neuromuscular development."

"The epileptic reaction from its mildest to its severest manifestation is really a protection, for it obliterates reality and reduces the subject to the lowest level of organic response, that of the comatose state. It dispels an intolerable demand and the epileptic retreats to a state of harmony and peace."

One wonders why a nocturnal epileptic in a fit of self-hatred should have to retreat from the harmony of slumber to that of coma. However, we are finally informed that these lucubrations "give us a more rational understanding of the nature and mechanism of the disorder itself and render the plan of handling and treating the essential disorder more understandable and practicable." Now, of course, they do nothing of the kind. On the contrary, jargon of this sort fails to throw even so much light as shone in Milton's Hell, for that at least made visible the darkness and caused the traveller to walk delicately therein.

It is a simpler task, however, to say what epilepsy is not, than what it is. It has been already described as an occasional paroxysmal discharge of a nerve center or group of centers occurring apart from volition and accompanied by interference with consciousness. Perhaps "impairment" might be a better word than "discharge," for many of the minor manifestations have such negative characteristics as to preclude the use of so positive a descriptive title. Even in the major seizure, the sequence of events can be most readily understood as a sudden cutting out of the highest level, the cortex, allowing the lower neuronic levels to pour downward an ungoverned stream of tonic postural impulses—in fact, an abrupt rigidity of the decerebrate type. The subsequent convulsions which have secured, by their dramatic quality and their greater duration, more attention than I think they deserve, probably represent the gradual return of cortical control, incapable, at first, of ordered volitional action. I suggested at the beginning of this paper that many diverse causative conditions must operate through a common mechanism to produce so constant a result, difference in the various manifestations of epilepsy, from the major for my claim is nothing less than a qualitative unity and quantitative seizure through *petit mal*, to migraine, to recurrent syncope and periodic endogenous emotional tempests. And in the gamut I should include, without equivocation, the voluminous mental states of unreality, and those prolonged and terrifying experiences known now as vasovagal attacks in which the patient is suddenly terror-stricken, suffers medi-

astinal oppression, has a feeling of tingling and swelling of the limbs, a sense of impending death and, losing precision of contact with his surroundings, experiences an acute transient seizure of cardiac hurry. Hughlings Jackson believed "idiopathic epilepsy to be far too difficult a subject for precise investigation unless we approach its consideration from the bases supplied by the principles deduced from the less complex kinds of cases." Probably the mechanism of the major attacks will be revealed by a study of the manifestations of the minor attacks which will be found, I think, to be fragments of the fully developed seizure—just as we have fragments of the decerebrate posture produced by imperfect mesencephalic block, so we may have fragments of the great fit produced when abnormal conditions, probably of a vascular nature, are not sufficiently violent to unseat the entire cortical function.

The vasovagal attack which I have just described, if condensed in point of time, would be the same as the phenomena of many of the petit mal attacks, and I can personally vouch for its identity with the sensations felt when, having compressed my carotids and vagi, I lost control of my experiment and, a few seconds later, of consciousness.

Leonard Hill, more intelligently and successfully—but, of course, less heroically!—compressed one carotid in himself: "the first effect was a sensation in the eye of the same side; then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm and down the leg. Finally, clonic spasms of the hand occurred, accompanied by an intense feeling of vertigo and alarm." Whether clonic movements occurred in my own case, I do not know, as I was alone at the time and have since lacked inclination to repeat the experiment; the sensation in the hands and forearms for the few seconds before loss of consciousness, was that of very powerful faradization; it was still present on my recovery but quickly passed away.

In connection with these considerations of vascular change, it is proper to remind ourselves of the cerebral phenomena produced by severe heartblock. Thomas Lewis finds that suspension of the mental functions is produced by a single period of asystole of from three to seven seconds' duration. In severer cases, where the pulse ceases for fifteen seconds or more, there occur cyanosis and stertor, and twitching of the face and limbs; fatal status epilepticus is by no means infrequent. In such cases there is a cessation of general circulation, but in the cats operated on by Stewart and Pike, cerebral and medullary circulation only were cut off. The immediate results were unconsciousness and bladder and rectal evacuation, severe tonic convulsions, then flaccidity (it must be remembered that midbrain circulation was obliterated); after the return of circulation there were clonic convulsions which pre-faced resumption of consciousness and complete recovery.

In the ability of the emotions to cause severe vasomotor fluctuations and, in certain persons, syncope, and in certain others, headache or minor and major convulsions, we have an approach to a psychologic mechanism in epilepsy, which differs as has already been suggested, from that impressionistically sketched by Pierce Clark and others.

One cannot leave the subject of vasomotor instability in epilepsy without mentioning the remarkable case published by Osler of Raynaud's disease of the fingers, feet, ears, and nose, occurring during six winters and accompanied only during such seasons by severe epileptic convulsion.

Such events raise again the question of the existence of motor nerves in the cerebral blood-vessels. These were thought for long to be absent, but Gaskill's observations have demonstrated their presence; the muscular walls in these vessels would seem evidence enough, but Biedl and Cushing have both made manifest that epinephrin blanches the brain as it does other tissues, and Wiggers has shown a definite diminution in the outflowing blood after the addition of epinephrin to the fluid circulating through the brain.

John Hartwell and I were once able to observe the brain of a patient during a general epileptic fit; the exposure of the parietal cortex had been carried out under local anesthesia whereby consciousness and normal reflex activity were retained up to the moment of the attack. The initial sign of the seizure was a sudden whitening of the cortex which was no sooner remarked on than it was replaced by tremendous venous engorgement with protrusion of the brain beyond the level of the bone defect. This alarming engorgement, in which the previously blanched cortex became beet color and many of its veins swelled to half the diameter of one's small finger, was coincident with the tonic stage of the attack and the period of general clonic convulsion. The cortex failed to recover its normal appearance during the remaining twenty minutes of exposure, though its blood-red color was gradually mitigated in that period. The engorgement was apparently a part of the diffused cyanosis consequent on the general tonic spasm; the initial chalky appearance, however, must have been due to a vascular constriction which, I believe, was the proper beginning of the cerebral seizure.

Cushing has shown that faradic stimulation of the cortex produces pallor of the gray matter and it might be well to bear his observation in mind when considering what exactly we mean by the spread of focal attack. Such spread is too slow to be explicable by the passage of a nerve impulse and might well be dependent on a quickly widening area of cerebral anemia. McRobert and Feinier advanced recently an interesting theory to account for the fact that generalized epileptic seizures result more often from neoplasms in the temporosphenoidal

lobes than from tumor formation in any other cerebral area. These observers believe that the topographical relation of the sylvian artery to these lobes renders the former susceptible to abrupt compression from the transient edemas surrounding the growth; such sudden devascularization, they believe, renders the cortical elements unstable, but as I have already suggested, the mechanism may be rather that the cortex is cut out by such an event and lower levels released by lack of adequate control.

Following up the notion that the initial tonic phase of the grand attack is due to a stream of ungoverned impulses from the lower neuron levels, we might remember that, in spinal convulsion due to strychnin poisoning, there is no initial tonic period and that clonic spasms occur from the beginning, whereas from midbrain intoxication by either tetanus or rabies, there occurs engravescent tonicity—lockjaw and risus sardonius—or tonic convulsion as the first manifestation of disease. It would appear that the discharges of energy produced by strychnin poisoning would be included in the definition of epilepsy advanced by Hughlings Jackson, as would indeed those associated with rigor, simple shivering or that curious somatic tremor which some people experience after micturition. It is for this reason that I have thought it necessary for clinical purposes to make it clear that we consider as medically epileptic only phenomena associated with interference with consciousness. It is not desirable to say that, in what Gowers called the borderland of epilepsy, there is impairment of consciousness, for in the voluminous mental state there is often a feeling of expanded receptivity, a subjective enlargement of the orbit of awareness, a sense of prescience. We must not deny the possible expansion of consciousness in such events merely on the grounds of their subjectivity—our sense avenues are the usual, but—in the light of the new physics—may not be the sole pathway of ethereal impulses! However, an extraordinary acuteness of reaction to sensory stimulation may occasionally be seen in epileptic persons. Convulsions could be produced without fail in two patients under my observation. In one, a child, by flicking or sharply tapping the left side of the nose, in the other, an adult, by rubbing the dorsum of the left foot. The attack in the first case consisted in immediate dilatation of the pupils, tonic spasm of the arms and chest, respiratory arrest, cyanosis and slight frothing at the mouth, the whole lasting sometimes fifteen, sometimes twenty seconds, and ending in flaccid exhaustion after return to consciousness. There were no clonic movements in this child whom Dr. Jackson was good enough to come to the hospital to see and whose fit he regarded as a typical bulbo pontine explosion.

In my second case, who was under the care of Dr. Farquhar Buzzard, the convulsions were general and followed the usual pro-

cedure of a major seizure. The right parietal cortex was exposed in this case without anything of note being revealed.

In 1920 there was admitted to the neurological department of Bellevue Hospital, a boy aged 17 years whose history stated that for twelve months he had been subject to instant falling to the ground on hearing a sudden sound—a horse pawing the ground in a quiet street, a boy whistling behind him—would be enough to produce cramp in the left leg and a quick fall in which he was always bruised. He denied any loss of consciousness and certainly to onlookers, gave for a long period, no evidence of such. However, after two years of these events, frequently repeated, he had a major convulsion which came on after the slamming of a door and was preceded, as were the other attacks, by a feeling of cramp in the leg. These larger seizures did not entirely replace the fallings after sudden sounds. I induced both types of attack with ease, but did so rather seldom as he would fall violently on his face and, so innocent was he of any reflex of defence, I feared for his safety.

This is an example of the reflex acusticomotor epilepsy described by Oppenheim; for many months, the boy was thought by some competent men to be hysterical. His first fall occurred at school when he was trying to solve a tough problem at the blackboard; his crayon broke and he fell sharply to the ground—a refuge, perhaps, from his embarrassment—indeed an admirable example of a sly subliminal mitigation of Fate! His infantile history, however, supplied an organic background if not an organic explanation. Three weeks after birth there was still present a very large caput succedaneum over the right occipitoparietal area. This was opened and a blood clot evacuated. There was said to have been a bone defect in this area at that time. Symptoms of meningitis are said to have been present during the first two weeks of life. When five months old he had a general convulsion; later, he began to have jerky movements when falling asleep and on waking; in his mother's words: "The left leg would come up and the left shoulder and body go forward; the movement would occur eight or ten times but varied in number and intensity." These hypnogogic events lasted with lessening severity for fourteen years; two or three years later they were replaced by the recurrent episodes just described.

The parietal cortex was exposed. A great thickening of the dura was discovered, produced by an old organized, subdural clot. The attacks have become rare since the operation, and hypersensitiveness to sounds uncommon. This boy always stood near the top of his class; he is now in his second university year, an athlete of splendid physical development, and his peculiarly sweet character scarcely coincides with the sonorous descriptions of ethical and emotional depravity described

by some as being both the cause and the result of maladies such as his. This case has been told at some length because of its intrinsic oddness, but also because, while refuting emotional torment as the cause of organic cerebral explosions, it shows how a brain can be sensitized by trauma and react quickly to stimulation either by primary discharge or in answer to rapid vasomotor changes.

It is needless to discuss here the exact relationship between migraine and epilepsy; they are both found in persons of neuropathic type and heritage: both diseases often occur in different persons of the same stock and very many instances are reported where they existed at different times in the same patient. In a case of my own, the left-sided headache was ushered in by a sensation of flashing lights in the right temporal field, numbness in the right hand, and ingravescent hemianopia, alexia, and aphasia, all of which became practically complete. The subsequent headache lasted for some hours during which the patient behaved rationally. Following the seizure he had complete amnesia for the period of the attack with the exception of its first half-hour.

Migraine has been reported as alternating in three patients with family periodic paralysis—but it would be futile to list here the numberless forms of equivalents which have been described and are entirely familiar to this audience. Earlier in this paper I used the term the spectrum of epilepsy. My general purpose has been to suggest that in this spectrum are many colors which merge insensibly into each other; uremia and eclampsia of pregnancy, the fits of general paralysis and brain tumor are there: the spasmophilic conditions of infancy are also to be thus grouped, including certain forms of tetany associated with confusion of consciousness: the chorea of pregnancy may be in this spectrum also, and—not very deep in the ultraviolet sector—are probably some cases of major hysteria, the motor phenomena of which suggest the temporary abandonment of a cortical for an infracortical mechanism.

Turner divided all these symptoms into: the organic epilepsies arising from trauma, and focal organic disease of the brain; the early epilepsies, those of childhood and infancy, often accompanied by infantile cerebral hemiplegia or mental retardation; the late epilepsies, emerging from chronic disease of the heart and blood vessels, or from intoxications by alcohol, lead or syphilis, and those springing from eclamptic conditions; lastly he sets down "idiopathic, essential, or genuine epilepsy, a malady arising about the period of puberty, usually progressive, difficult or impossible of arrest and fathered by no tangible or ascertainable cause." I submit that while all these maladies most properly are called epileptic, their arbitrary subdivision gives an air of unlikeness to their descriptions, incompatible with fact and that there is no ground for defining "genuine epilepsy" as an isolated group except that in the

other groups, causative factors have already been discovered. Analysis has been almost overdone; synthesis of these symptoms displays their essential unity. Idiopathic tetanus has only recently been obliterated by the discovery of the cause of the disease; and to this was added shortly, its prevention, mitigation and cure. It is that the same may happen to idiopathic epilepsy that we labor today.

SYMPTOMATIC EPILEPSY IN BIRDS

AN ANATOMICO-PATHOLOGIC AND CLINICAL REPORT

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From the literature of comparative zoopathology it seems that epileptiform convulsions due to lesions of the brain are rather frequent in birds. Yet the pathologic findings have not always been studied. For this reason, I am reporting the case of a chicken which for several months had shown convulsive attacks, of jacksonian type, in the right wing.

REPORT OF CASE

Clinical History.—The chicken of medium size, in April, 1918, began to show a constant tendency to fall toward the right. Sometimes it would fall and lie on this side; it would then rise again, walk fifteen or twenty steps and fall again. Later it began to be attacked with clonic spasms of the right wing and foot, with a tendency to rotate the head toward the right, and frothing at the mouth. The attack would last from fifteen to twenty minutes and leave the animal somewhat confused. During August, about four months after the onset, the convulsive attacks became more frequent, and occasionally occurred several times a day. They were always of the same type. Towards the end, the chicken became unable to walk; it would try to elevate the right leg, but at every effort would fall, always to the right. To advance, it would lean against the wall for support on the right side. The chicken always lay on this side, and invariably rotated the eyes downward at intervals. Finally, as the convulsive attacks became more frequent, the bird was killed on September 17, 1918, five months after the onset of the symptoms.

Necropsy and Microscopic Examination.—On the ventral surface of the right half of the pons (Fig. 1) was an irregular depression two millimeters in diameter which, to palpation, gave an impression as if there were very hard tissue beneath the surface.

The cerebrospinal axis was hardened first in formaldehyd and later in Müller's fluid; it was then embedded in celloidin and cut in frontal serial sections from the proximal extremity of the prefrontal lobe to the conus medullaris. The sections were stained by the Weigert-Pal or the Pal-fuchsin method. No lesions were found anterior to the point of exit of the seventh pair of cranial nerves.

In a transverse section at the exit of the seventh pair of nerves, the area corresponding to the right tractus tectobulbaris cruciatus in its ventromedial portion, was partly decolorized. The specimen also revealed the disappearance of some fibers of the trapezoid body, mainly on the right side; the most dorsal fibers of this body remained intact. In a frontal section of the pons in a plane distal to the preceding one, the area of the tractus tectobulbaris was degenerated, especially on the right side.

The transverse sections of the pons (Fig. 2) at the level of the abducens nucleus, contain a focus which appears as a small mass of black pigment (melanin) of circular shape, and occupies the right ventromedial region of the pons. The fibers of the tractus tectobulbospinalis cruciatus are greatly

rarefied on the right, and much less so on the left. The ventral radicular fibers of the sixth nerve are also involved in the disease focus on the right side.

A frontal section at the proximal end of the medulla oblongata (Fig. 3) reveals an area, corresponding to the ventromedial portion of the right side of the pons, which is replaced by a substance, chocolate brown in color externally, and yellowish internally, which contains fat globules, fragments of myelin fibers, and hematoidin crystals (broken down blood clot). The borders of this focus are irregular and serrated, and the surrounding tissue has lost its nerve fibers. The focus has destroyed all fibers of the right tractus tectobulbaris cruciatus up to the neighboring fibers of the tractus tectospinalis, and extends, beyond the raphe, into the extreme ventral portion of the homologous region of the left side.

Before discussing the clinical features of my case, it may be well to mention the little that has been published on the subject. Marek¹ simply states that birds are subject to epileptic attacks. Friedberger and Frohner² say that epilepsy is not rare in birds, especially parrots, canaries and chickens. Friedberger particularly describes a convulsive



Fig. 1.—Ventral aspect of the brain of the epileptic chicken. The right half of the pons is depressed and transformed into a dark tissue of cicatricial appearance.

attack in a chaffinch. During the attack this animal shook, lost consciousness and had violent clonic spasms of the muscles of the extremities; it fell on its back and remained in one place with legs and wings extended, and with the head drawn backwards; the beak and eyelids were successively opened and closed, and the eyeballs were rotated. Finally, consciousness was rapidly restored. Marek says that birds in convulsive attacks, first emit loud cries, shake, and fall on one side, or on the back; the feet then jerk rapidly, the eyes are strongly rotated, the head is drawn to one side, and the beak opens and closes. Exceptionally, there is shaking of the whole body. The convulsions disappear after one or two minutes, and the animal remains stupefied for some

1. Marek: Hirnblutung bei einem Kanarienvogel. *Allatorvosi lapok*, Budapest **31**, 1908; cited in *Ellenberger Sch. Jahrsb. d. vet. Med.* 1908, p. 363.

2. Friedberger und Frohner: *Lehrbuch der spez. Path. und Ther. der Haustiere*, Stuttgart, Enke, 1904, p. 737.

time. It then rises, wanders about for a while, and supports itself with its beak and extended wings.

Reimann and Beyer³ describe a cock which was frequently attacked with convulsions of the legs. It would fall and rise, and then finally rise again and eat with appetite. The necropsy disclosed dilatation of the heart and atrophy of the walls of its ventricles. (The brain was not described). Bruno⁴ mentions a canary which showed unilateral paralysis, and died from cerebral hemorrhage. This was confirmed by necropsy. Sustmann⁵ mentions epileptiform attacks in a gray parrot. Reimann⁶ reports a cock affected with inflammation of the joints of

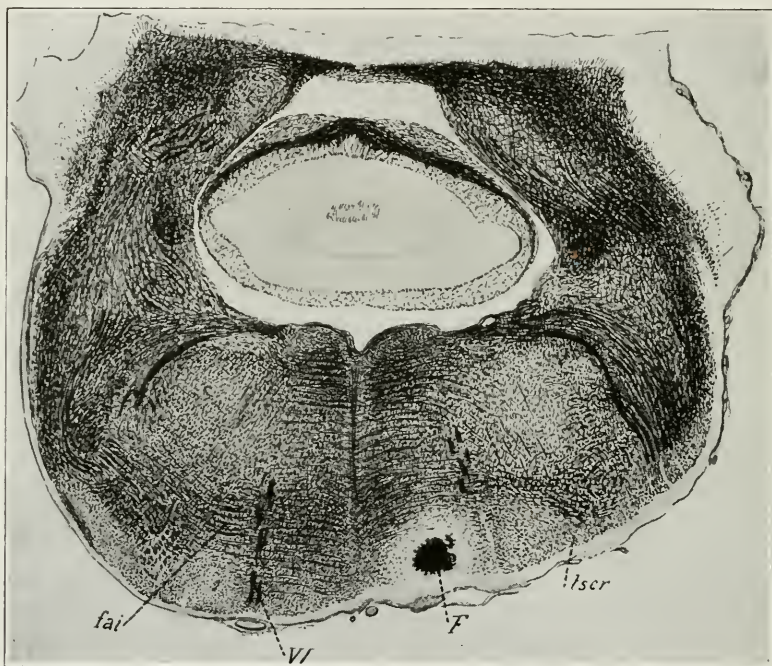


Fig. 2.—Transverse microscopic section of the pons at the proximal pole of the hemorrhagic focus. F, hemorrhagic focus; tb, tractus bulbospinalis cruciatus; tb, bulbar portion; tscr, spinal portion; VI, abducens nucleus; fai, internal arcuate fibers.

3. Reimann und Beyer: *Krankheits und Sektionsbefunde*, Dresdner Bl. f. Geflügelzucht, 1888; cited in *Ellenberger Sch. Jahrsb. d. vet. Med.*, 1889, p. 187.

4. Bruno: *Ueber Hirnblutungen bei Kanarienvogeln*, Gior. d. r. Soc. d. Accad. vet., 1902, p. 513; cited in *Ellenberger Sch. Jahrsb. d. vet. Med.*

5. Sustmann: *Epileptiforme Anfälle bei einem Graupapagai*, Münch. tierarztl. Wehnschr. **66**:121, 1915.

6. Reimann: *Krankheits und Sectionsgeschichte*, Dresdner Bl. f. Geflügelzucht, 1886; cited in *Ellenberger Sch. Jahrsb. d. vet. Med.*, 1887, p. 139.

a leg caused by mechanical injuries, which developed, almost suddenly, in addition to the local disturbances, a peculiar nervous syndrome. The animal drew the injured leg up to the abdomen, and kept it well stretched out behind him and extended upward; the bird then again retracted the leg on the abdomen, and followed this by touching the foot to the floor with quick jumping jerks, and finally started to use it properly. No necropsy was performed. The same author states that, in cerebral hemorrhage in birds, retropulsion and disordered movement have been observed. The necropsy made by him on a cock that had been walking backwards and turning to the left around itself with head rotated, revealed hemorrhages into the cerebellum and pons, besides acute hyperemia and edema of the right half of the brain.

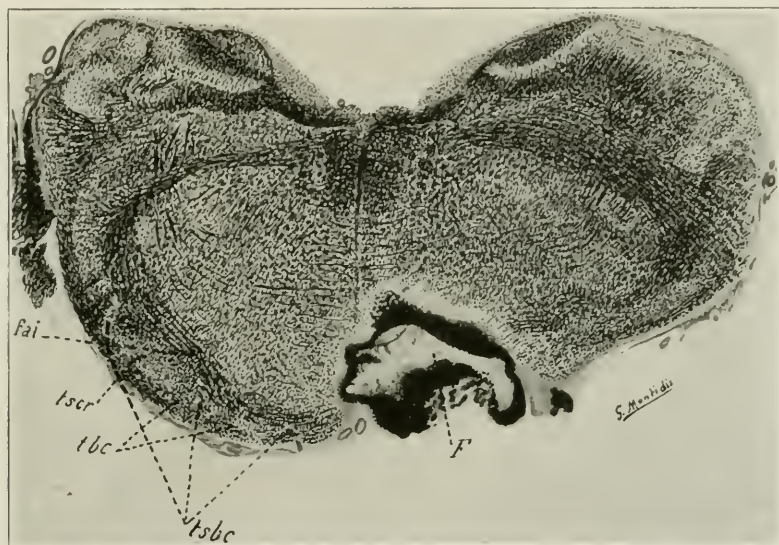


Fig. 3.—Transverse microscopic section of the pons at the level of the abducens nucleus where the hemorrhagic focus had reached its greatest extent. The focus destroys all the fibers of the tractus tectobulbaris cruciatus and part of those of the tractus tectospinalis. F, hemorrhagic focus; tbc, tractus bulbo-spinalis cruciatus (tb, bulbar portion; tscr, spinal portion); fai, internal arcuate fibers.

Marek mentions the case of a canary which, in its fits, rotated the head obliquely toward the left, and moved around in a circle toward the right, with the left wing down. There was found at the necropsy an extravasation of blood in the left pes pedunculi which corresponded to a frontal section made between the corpora quadrigemina and the nucleus of the sixth nerve.

In the chicken which I examined, convulsive attacks, distinctly unilateral, of the wing and leg occurred during several months, and

were followed by progressive paresis of the same parts. The necropsy disclosed a hemorrhage in the ventromedial portion of the pons.

Birds may, therefore, have the syndrome of essential epilepsy; general convulsions of classical epileptic type, which involve the eyes, beak, head, wings, and legs. Observations of lesions of the brain in such cases are as yet lacking. In addition, birds may have attacks of symptomatic epilepsy, which result from lesions of the cerebral hemispheres or of the basal portion of the pons. My case here cited, is an example of the facility with which epileptiform attacks are developed in chickens in consequence of a hemorrhage into the pons.

In man, spasms due to lesions of the pons, have been observed shortly after the inception of a rapid lesion (abscess, hemorrhage), and after a certain time in slowly progressive lesions, such as tumor. Such attacks are more or less transitory and affect muscles of one side which are already paretic; or else they consist of twitching and spasm of the face, and rotation of the eyes in different directions (Monakow). Unilateral convulsions which generally affect the opposite half of the body have often been observed: more frequently there occur general convulsions. It has not yet been determined what particular structure of the pons, upon irritation, gives rise to these attacks. Nothnagel believed it possible that general motor irritative manifestations might arise from the pons alone; whereas Bechterew held that spasms in pontine lesions depend on indirect irritation of the cerebral hemispheres in the Rolandic area. In my chicken the symptoms began with paresis of the right wing, foot and abducens nerve, to which were added, either simultaneously or soon after, convulsive attacks of a clonic type in the wing and foot on the same side. Microscopic examination revealed a hemorrhagic focus in the distal portion of the pons, which destroyed completely the bulbar part of the right tractus tectobulbospinalis cruciatus and partly destroyed its spinal portion. The lesion extended to the left beyond the raphe.

As is well known, voluntary motion, so-called, is subserved in birds by the tractus tectobulbaris and tractus tectospinalis; the latter, according to Edinger⁷ occupies the lateral portion of the lateral periphery of the mesencephalon; hence, in all probability, it is located in the corresponding position in the pons. Now, as the fibers of the tractus tectobulbaris were in my case almost all destroyed, and those of the tectospinalis were badly damaged, we can understand why there were at times symptoms of permanent deficiency and at other times evidences of irritative phenomena. Human pathology offers numerous similar examples, especially following destructive focal lesions in the

7. Edinger: Vorlesungen über den Bau der nervösen Centralorgane des Menschen und der Thiere, Leipzig, F. C. W. Vogel, 1911, Vol. 2, Fig. 88.

rolandic region or in the pes pedunculi. Comparative anatomy has also taught us that the complex of bundles of the above named tracts decussates mostly in the mesencephalon and then passes along the ventral margin of the pons. It is therefore easy to understand why symptoms of irritation and of deficiency, caused by partial lesions of these tracts on the right side of the pons, were present in the extremities of the same side. The tendency to the formation of a cyst and the development of cicatricial tissue, explains why the parietic-irritative syndrome had become more marked during the last months. This case, which meets the requirements of an experiment, demonstrated that, at least in birds, the tractus septomesencephalicus cannot be considered identical with the tractus corticospinalis from the functional point of view, as some believe; but instead, that the tractus tectospinalis is to be considered as equivalent to the tractus corticospinalis of the higher mammalia, since irritation of the latter will give rise to convulsive attacks of a clonic nature, with at the same time paresis of one side. Therefore we may conclude that these physiologic properties of the tectobulbar and tectospinal tracts have been transported by phylogenetic evolution into the neophyletic motor tracts (tractus corticospinalis). They come, not from the tectum as do the paleophyletic ones, but from the cortex cerebri, and have developed beneath the first, which, in birds, pass along the ventral border of the trunk of the encephalon.

INFANTILE PROGRESSIVE MUSCULAR ATROPHY OF WERDNIG-HOFFMANN TYPE

CASE REPORT WITH NECROPSY *

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MINNEAPOLIS

Twenty-one years ago Werdnig¹ published the first description of the clinical and necropsy findings of the syndrome which has since borne his name. Two years later Hoffmann² described the pathology of one case, gave the clinical notes of three others, and confirmed Werdnig's conclusions. The literature at present contains studies of about fourteen typical, non-congenital cases, only six with histopathologic findings. The first cases recorded in the American literature were those of Bliss in 1916.

In 1900, Oppenheim³ described a syndrome, now known as amyotonia congenita, which he observed was not hereditary nor familial but rather congenital; a tendency toward improvement was characteristic, and the seat of the principal pathology was thought by him to be in the muscles. Subsequent to Oppenheim, Spiller,⁴ Lereboullet and Baudouin,⁵ and Councilman and Dunn⁶ reported necropsy observations confirming those of Oppenheim. Spiller and Lereboullet and Baudouin, however, found only slight changes in the muscles, and the spinal cord to be practically normal. Thus anatomic studies in amyotonia congenita revealed lesions similar to those found by Werdnig and Hoffmann.

Holmes⁷ reports a case of amyotonia congenita and emphasizes the similarity between the pathologic findings in the familial, progressive, non-congenital form (Werdnig-Hoffmann) and those of amyotonia congenita. Krabbe⁸ reports six cases of congenital and familial

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* Read at the annual meeting of the Central Neuro-Psychiatric Association, Rochester, Minn., Oct. 21, 1922.

1. Werdnig: Arch. f. Psychiat. **22**:706, 1891.

2. Hoffmann: Deutsch. Ztschr. f. Nervenhe. **3**:427, 1893.

3. Oppenheim: Monatschr. f. Psychiat. u. Neurol. **8**:232, 1900.

4. Spiller: Univ. Penn. M. Bull. **17**:342, 1904.

5. Lereboullet and Baudouin: Bull. et mêm. Soc. méd. d. hôp. de Paris **36**:1162, 1909.

6. Councilman and Dunn: Am. J. Dis. Child. **2**:354 (March) 1911.

7. Holmes: Am. J. Dis. Child. **20**:405 (Nov.) 1920.

8. Krabbe: Brain **43**:166 (July) 1920.

spinal muscular atrophy and one of the hereditary, non-congenital, progressive type, all with similar clinical and pathologic findings. He concludes that there are two types of amyotonia congenita: one is the non-hereditary, favorable variety with muscular hypotonia but no definite atrophy; the other shows atrophy of the anterior horn cells and muscles, and usually has a positive hereditary and familial history. Huenekens and Bell⁹ report a case of the Werdnig-Hoffmann type with a positive hereditary history, as well as the familial history that a previous child probably suffered from amyotonia congenita. They give a list of typical and atypical cases. It would seem that their atypical cases could properly be classed under the second group of the grave congenital types of Krabbe. Reuben¹⁰ has summarized the pathologic findings in cases of amyotonia congenita which had been reported up to April 1917. Barton¹¹ has reported a familial noncongenital case with necropsy. Duken and Weingartner¹² submit a complete case report with familial and congenital history. The three cases of Knox and Powers,¹³ in which necropsies were not performed, belong in the congenital category. The report of Leenhardt and Sentis¹⁴ leaves one in doubt about their first case; their second case apparently is not congenital. In neither case was there a necropsy.

Thus it appears that the main clinical distinctions between amyotonia congenita and the disease of Werdnig-Hoffmann are: (1) the hereditary, familial or congenital origin; (2) the presence or absence of atrophy; (3) the course of the disease, as regards its tendency toward progression or recovery. The relation of the necropsy findings to the clinical picture is not definite, and it is obvious that a clear understanding will not be reached until more cases with histopathologic findings are reported.

REPORT OF A CASE

History.—Annie M. was admitted on March 14, 1921, to the out-patient service of Dr. Bronson Crothers at the Children's Hospital. She was born at full term on Jan. 15, 1921, and was the sixth child of healthy, Italian parents. Delivery was easy and natural, and the child was breast-fed. Muscular activity during the first month of life was normal. The child gained in weight and was apparently well until the age of one month. During the second month, weakness in arms and legs had been progressive.

Family History.—The first child in the family is well and aged 6 years and 9 months. The second, third, fourth and fifth children died at the ages of 3, 4, 5, and 4 months, respectively, all from pneumonia. All had a period of

9. Huenekens and Bell: *Am. J. Dis. Child.* **20**:496 (Dec.) 1920.

10. Reuben: *Arch. Int. Med.* **20** (Nov.) 1917.

11. Barton: *Lancet* **2**:922 (Nov. 22) 1919.

12. Duken and Weingartner: *Ztschr. f. Kinderh.* **29**:245 (July) 1921.

13. Knox and Powers: *South. M. J.* **13**:86 (Feb.) 1920.

14. Leenhardt and Sentis: *Arch. de méd. d. enf.* **24**:137 (March) 1921.

"paralysis" before death, similar to that of the case here reported. In other words, the familial factor in this case is conspicuous. No further details of the history were obtainable.

Examination.—A normally developed, plump baby, aged 2 months, who was apparently comfortable. She gave a weak cry at times. The head was of normal shape, and the anterior fontanel was open, though there was no bulging. The cranial nerves were normal. The arms and legs were moved very little, though the limbs on the right moved more than those on the left. The toes flexed on stimulation but there was no motion at the hips or the knees. The arms moved at the elbows, wrists, and fingers. The knee and ankle jerks were absent. There was no Babinski sign. No definite atrophy, group paralysis, spasticity, or contractures were present. The abdominal muscles and the external muscles of respiration were all weak. Breathing was diaphragmatic in type. The electrical reactions were normal with a fairly strong faradic current and with a galvanic current of 5 milliamperes. Sensation was normal and the results of further physical examination were negative. No Wassermann test was made. The spinal fluid was not examined. A roentgenogram revealed a heavy fat layer, and apparent atrophy of the muscles of the arms and legs.

Course.—March 23: The right arm flexed easily at the elbow, the left did not flex so easily. The fingers of both hands flexed and extended fairly well. The legs were flaccid, and motionless except for the occurrence of plantar flexion of the toes. It was very obvious that muscle tone had diminished since March 14. A reaction of degeneration was now present. No reaction was obtained in the rectus abdominis muscles.

April 4: Weakness of the forearm muscles was quite marked on both sides. Movement of the toes was weaker.

April 18: Reaction of degeneration and weakness was further advanced. A pulmonic infection occurred during the last days of April and the first part of May.

June 6: Reaction of degeneration was complete in arm, leg, and trunk muscles. A roentgenogram of the trunk revealed normal viscera and spine. After this visit to the out-patient department, the patient again developed a pneumonic affection and death occurred June 23, 1921, seven days after the last hospital examination.

Fibrillary twitchings were not noted at any time. The diaphragm was not involved, and the facial muscles did not give the reaction of degeneration. As the child was not an in-patient of the hospital, the study of the case was not satisfactory.

Necropsy.—A necropsy, performed by Dr. Cobb twelve hours after death, was restricted to an incision over the spine. The length of the body was 95 cm. The abdomen was protuberant; the thorax was funnel shaped and small, with very thin muscles. The pectoral muscles were particularly small and soft. The buttocks were normal. The pupils were round and equal, and measured 3 mm. in diameter. The hair was thin, and there were brown, scaling macules on the scalp. The anterior fontanel measured 3.5 by 3 cm. and was depressed. There was no discharge from the ears or from the nose. The genitals were normal. There was an unusual configuration of the anus; a band below caused the anus to point almost straight backward. The subcutaneous fat layer over the back was about 7 mm. in thickness. The muscles were pale and soft, and of fibrous appearance and consistency. Specimens were taken which included: (1) A thin sheet of the latissimus dorsi. (2) The

right brachial plexus. (3) The right hamstring muscle. (4) The left sciatic nerve. (5) The spinal cord and most of the medulla. The only gross abnormality of the cord or membranes, was a slightly purplish discoloration of the dura mater over the lumbar enlargement. Spinal fluid was taken by cistern puncture. The thymus, adrenals, viscera, brain and other muscles and nerves could not be examined.

Examination of the spinal fluid gave negative results with Wassermann and globulin tests.

Histopathology.—Sections of the medulla, below the lower portion of the vestibular nucleus, reveal no changes in the nuclei of the ninth, tenth, and



Fig. 1.—Thoracic segment of spinal cord showing normal myelin in dorsal roots but partial degeneration of ventral roots. Van Heuman stain.

twelfth cranial nerves. Root fibers of these nerves are normal. There are no connective tissue nor glial abnormalities. In the cervical region of the cord (cresyl violet stain) the total number of cells is reduced. In one section there are three normal anterior horn cells on one side and five on the other. The cells which remain are reduced in size. They show signs of chronic degeneration; the chromophil substance is closely packed, and the cells appear small and dark. Cells in the dorsal horn are normal. The Marchi stain reveals no acute degeneration of the myelin. Phosphotungstic acid stains show no altera-

tions in the neuroglia. The ventral nerve roots, within the white matter of the cord, as well as outside of the cord substance, show, by Weigert's method, marked diminution of myelin. The contrast between these and the normally stained dorsal roots, is quite pronounced. Other structures are not involved.

One section from the thoracic region of the cord contains but two normal nerve cells in one ventral horn and four in the other. The remaining cells are attenuated and show chronic changes similar to those in the cervical sections, with the exception that one cell has swollen, pale cytoplasm, and an eccentric

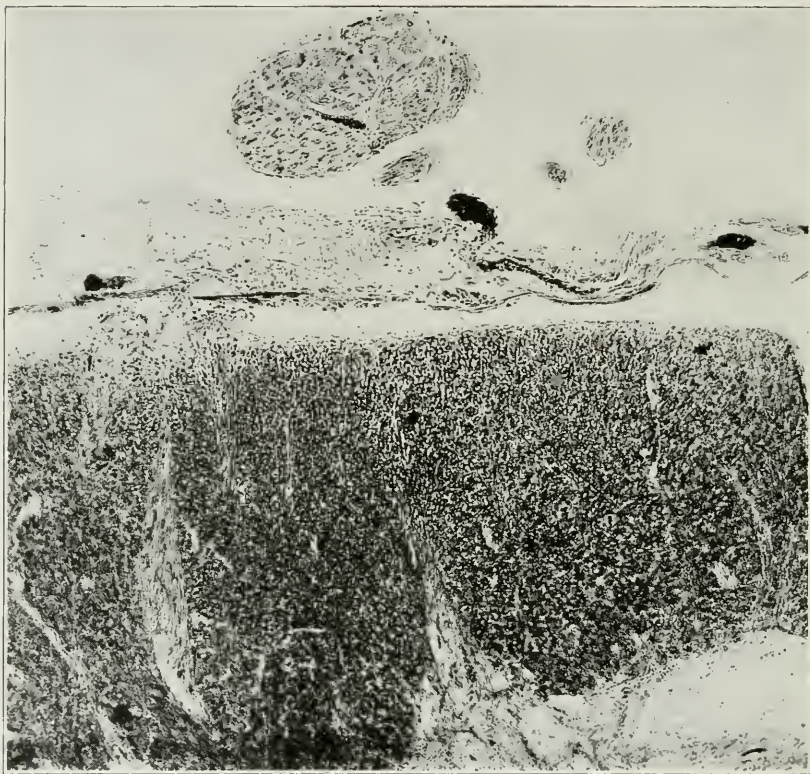


Fig. 2.—Ventral root of lumbar segment of spinal cord showing degeneration, as evidenced by lack of myelin. Van Heuman stain.

nucleus, a condition of acute degeneration. The cells in Clarke's column and the dorsal horns are normal. Van Heuman's¹⁵ stain shows marked diminution of myelin in the ventral nerve roots (Fig. 1). The Marchi method demonstrates phagocytes in the blood vessels, laden with darkly staining bodies which are evidently fat particles. No other changes are shown by this method. Phosphotungstic acid slides are negative. In the lumbar region of the cord there is marked diminution in the number of ventral horn cells; about one third of those which remain in one ventral horn are normal, and in the other ventral horn one

15. Van Heuman, Luden: *Centralbl. f. allg. Pathol.* **23**:98, 1912.

fifth are unchanged. The chronic cell changes described above are prominent. Acute changes are demonstrable in smaller numbers. All other parts of the section, except the ventral nerve roots, are normal. Weigert and Van Heuman stains reveal striking diminution of myelin in the ventral roots (Fig. 2). The phosphotungstic acid and Marchi stains give negative results. Cross sections of parts of the brachial plexus stained by Van Heuman's method, show many nerve bundles devoid of myelin. Other bundles have only little normal myelin. The blood vessels and supporting tissues are not altered. The myelin changes are

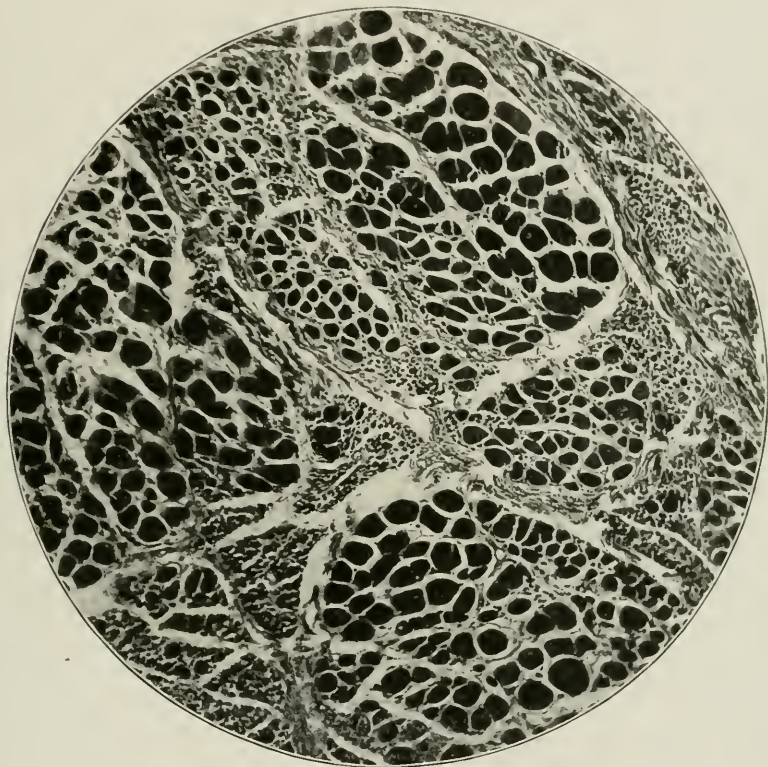


Fig. 3.—Latissimus dorsi muscle showing small muscle fibers, with increase of connective tissue about some individual fibers, and a relative increase of connective tissue throughout the whole area. Mallory's connective tissue stain.

also present in a terminal nerve within the hamstring muscle. Cross section of the left sciatic nerve shows changes like those of the brachial plexus. Cells of the neurilemma sheaths are not changed.

Sections of the latissimus dorsi (Fig. 3), hamstring and erector spinae muscles show marked atrophy of many fibers, but the small fibers appear to be fetal in type rather than degenerated. Normal fibers may appear in atrophied fasciculi, or whole bundles of fibers may be normal. Frequently a whole bundle is atrophied. Some of the smaller fibers have the appearance of smooth muscle; cross striations are lacking. The picture is not that of secondary atrophy.

There is marked, relative increase of connective tissue, but only slight true fibrosis. This is evidenced by an occasional muscle fiber, surrounded by a fine network of connective tissue. No fatty changes are noted.

COMMENT

The similarity of these pathologic findings to those noted in the "second form of amyotonia congenita" of Krabbe, and in the familial type of Werdnig-Hoffmann, would indicate that these two syndromes should be regarded as a single disease entity. Whether symptoms appear before birth, as suggested by the absence of intra-uterine movements, or whether they do not present themselves even before the second half-year of life, would seem an insufficient criterion for the separation of the two syndromes, since the myelopathic processes do not seem to be distinguishable. The amyotonia congenita of Oppenheim, however, (with its pathologic changes limited to the muscles, and with a favorable prognosis) as originally described, should be regarded as a separate and more restricted syndrome.

CYSTIC BLASTOMYCOSIS OF THE CEREBRAL GRAY MATTER

CAUSED BY *TORULA HISTOLYTICA* STODDARD AND CUTLER *

WALTER FREEMAN, M.D., AND FRED. D. WEIDMAN, M.D.

PHILADELPHIA

The diffuse form of blastomycosis of the central nervous system appears as plastic meningitis which may be combined with cystic infiltration of the gray matter. The central nervous system is involved chiefly, and sometimes apparently primarily, and the causative organisms are found in the meninges and in the cysts.

REPORT OF A CASE

History.—H. P., a man, aged 39, was under the care of Drs. Charles H. Frazier and William G. Spiller, at the University Hospital from May to July, 1921. On admission he complained of severe paroxysmal headache of one month's duration. The headaches were progressive and were not localized. Projectile vomiting had occurred daily during the week preceding admission. The eyesight had become poor during the preceding two weeks. He was aware of no loss of memory, aphasia, weakness or difficulty in walking, and had had no convulsions. A short time previously he had had temporary difficulty in writing his name and in talking clearly.

Six months before admission a small mass had appeared on the right eyelid. This was diagnosed by a dermatologist as epithelioma, and disappeared under radium application. In 1911, his tonsils were removed and later some cervical lymph nodes became enlarged. In 1916 one of these was excised and after microscopic examination was pronounced to be Hodgkin's disease. The cervical nodes diminished in size under radiation but at the time of admission the patient had a large discrete mass in the right axilla.

Examination.—The patient was talkative, and somewhat facetious. On examination there were disclosed: moderate rigidity of the neck, nystagmus in looking to the left, slight exaggeration of the tendon reflexes and a positive Kernig sign. The optic papillas were elevated five diopters. There were no localizing signs, and in view of the negative findings in other organs a tentative diagnosis of unlocalized brain tumor was made.

The spinal fluid escaped under a pressure of 55 mm. of mercury; it was clear and formed a pellicle on standing. The cell count was 315. Thirty per cent. of the cells were typical lymphocytes, 6 per cent. were leukocytes. The remaining cells were round or oval with greenish, highly refractile bodies and a few intracellular granules which did not resemble nuclei. Many presented a double contour, and some were obviously budding. Smear preparations were not satisfactory.

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Course.—During the two months after admission to the hospital the patient became gradually worse. The headache became constant; left ophthalmoplegia developed; and, a few days before death, there were several generalized convulsions which were followed by weakness, loss of tendon reflexes and stupor. The temperature ranged between 99 and 100 most of the time, and did not rise above 101. The blood count, blood culture, urinalysis and Wassermann tests were all negative. The blood serum did not agglutinate the organism that was found, and was too anticomplementary to be used for complement fixation tests. Lumbar punctures relieved the headaches.

Necropsy Record.—A necropsy was performed eight hours after death by Dr. Balduin Lucké. External examination was essentially negative. The lungs showed hypostatic congestion but there was no pleuritis or consolidation. The heart was slightly enlarged and flaccid. The alimentary tract and pancreas were normal; the liver presented signs of an early atrophic fibrosis, and the kidneys showed marked tubular nephrosis with degeneration. Enlarged lymph nodes were found in the right axilla and in the mesentery, the largest of which measured 9 by 5 by 2.5 cm. The spleen was also enlarged. Both grossly and microscopically the glands presented an unusual picture which was characterized by loss of architecture, enlargement of the follicles, and infiltration with large, deeply staining cells which contained one or more nuclei.

The coincident enlargement of the spleen and lymph nodes, with a history of ten years' duration, suggested Hodgkin's disease, but the characteristics of the nodes, both grossly and microscopically, differed from the description given by Reed. The larger cells were too deeply stained, fibrosis was practically absent, and there were but few eosinophils. No yeast organisms were found in the sections but by means of guinea-pig inoculation they were recovered from the gland at the head of the pancreas.

The calvarium and cerebral dura showed no abnormalities. The fluid in the basal cistern and ventricles was cloudy, and contained numerous blastomyces. The brain was large and soft. The pia was not injected but was moderately thickened and edematous. A few minute pearly gray nodules were found over the convexity and in the great longitudinal fissure. *No cysts were seen until after formaldehyd fixation.* No areas of softening or tumor formation were detected by the palpating finger. After formaldehyd fixation the pia appeared thick and opaque. It stripped very readily, and then exposed a cerebral cortex in which cysts were so numerous at some places as to resemble soapsuds. These minute vesicles were raised slightly above the surrounding surface, and were sharply differentiated by the transparency of the contents. On section, the contained material did not flow but was of mucoid consistency. The external walls of some cysts were extremely thin but none appeared to have ruptured. Section through the brain revealed an extraordinary number of small cysts, some were almost a centimeter in diameter, whereas, others approached the limit of visibility. They were usually multilocular but always rounded. They were limited to the gray matter of the cortex and basal ganglions. The soapsuds appearance was also present in the cerebellum. The brain stem and spinal cord appeared normal. The spinal pia-arachnoid was slightly more thickened than the cerebral.

No focus of infection was found in the middle ear, accessory sinuses, or nasal or pharyngeal cavities.

Microscopic Examination.—The pia-arachnoid was considerably thickened by edema and fibrous tissue hyperplasia, together with cellular infiltration. Some

of the cells were lymphocytes and plasma cells, but most were large cells with faintly granular cytoplasm, and a large vesicular nucleus which was sometimes multiple. These cells formed collars about the pial vessels and extended out into the meshes of the tissue; some contained blastomyces. A few yeast cells lay free in the meshes of the tissue. No encysted or mycelial forms, and very few budding forms, were seen. No polymorphonuclear cells were present.

The outstanding abnormality of the cortex was the presence of the cysts, the great multitude of which gave rise to the soapsuds appearance mentioned in the gross description. They were scattered very irregularly; sometimes large ones were present at the surface and small ones beneath, sometimes the reverse arrangement existed. Large superficial cysts sometimes overlay one or two of smaller size. All, except the very minute ones, appeared to be multilocular and the partitions consisted of cerebral tissue (Figs. 1 and 2). Some cysts were well filled with organisms, phagocytic cells, and detritus; in others the fluid portion greatly exceeded the formed portion, and the cells were present only at the boundaries. The cysts appeared to form by internal expansion, which was due to the growth and multiplication of the organisms and the exudation of fluid, rather than to liquefaction necrosis. The cavities were sharply defined in almost all instances, though no special lining was present. Extension took place by direct progression, and by infiltration along perivascular sheaths. Many of these sheaths were dilated and contained phagocytic giant cells and yeast cells. There were no cysts in the subcortical white matter, though some of the perivascular sheaths were dilated and contained a few yeast cells and phagocytes (Fig. 3).

Giant cells were derived from the vascular adventitia. The cells became larger and more rounded, their nuclei dividing by fission. The larger cells took up organisms. Within the cells, these blastomyces developed capsules and distended the cells to very large dimensions—from 50 to 70 microns in diameter. The cytoplasm was disrupted, the nuclei were displaced, and the outline became irregular and indefinite. Finally the cell walls appeared to rupture and the organisms were set free in the cavity.

The nervous tissue immediately surrounding the cysts was compressed to some extent, the ganglion cells were elongated and flattened, their processes were distorted, and in the septa they were displaced toward one another. Generalized distortion of the cyto-architecture was not seen, except close to the cysts. Chromatolysis was general, and a few cells were undergoing neurophagia and other forms of destruction; but, except in the immediate vicinity of the cysts the cells appeared relatively normal. Fine nerve fibers were found in the trabeculae and cyst walls. Stains for fat showed that a small number of cells about and within the cysts, and along the perivascular spaces, contained fatty material. There was no notable increase in neuroglia, nor infiltration of the chronic inflammatory type. There was very little blood pigment. The yeast cells were concentrated in the cysts and perivascular spaces, and were not found in the intervals. The walls of the vessels appeared normal; no organisms were found in their lumens.

In the basal ganglions, the cysts were mostly of small size and were situated at a distance from the ventricle. A similar cystic condition was present in the cortex of the cerebellum. Here the cysts were smaller, but more numerous, especially in the superior vermis.

The brain-stem contained many minute cysts in the gray matter around the aqueduct of Sylvius and in the floor of the fourth ventricle. A focus was found in the left oculomotor nucleus that accounted for the ophthalmoplegia which

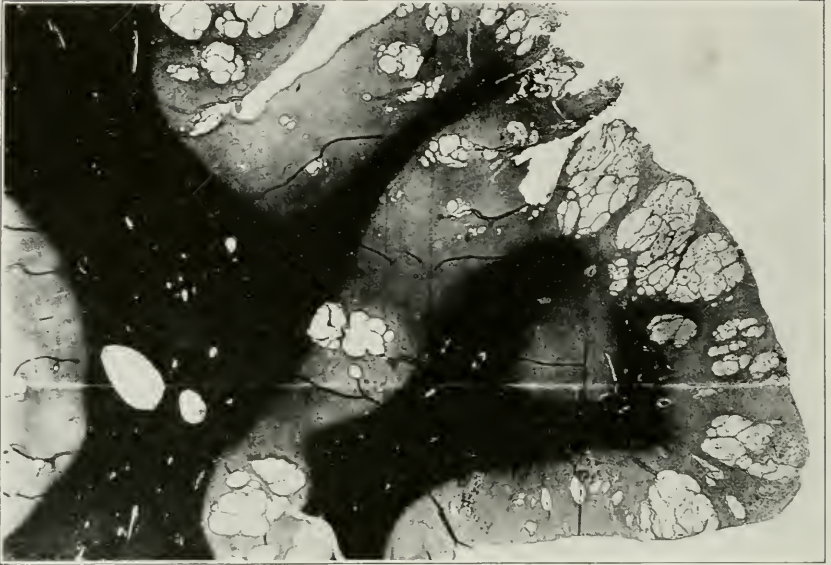


Fig. 1.—Multilocular cysts, limited to the cortex, in the frontal lobe. The pia has been stripped.

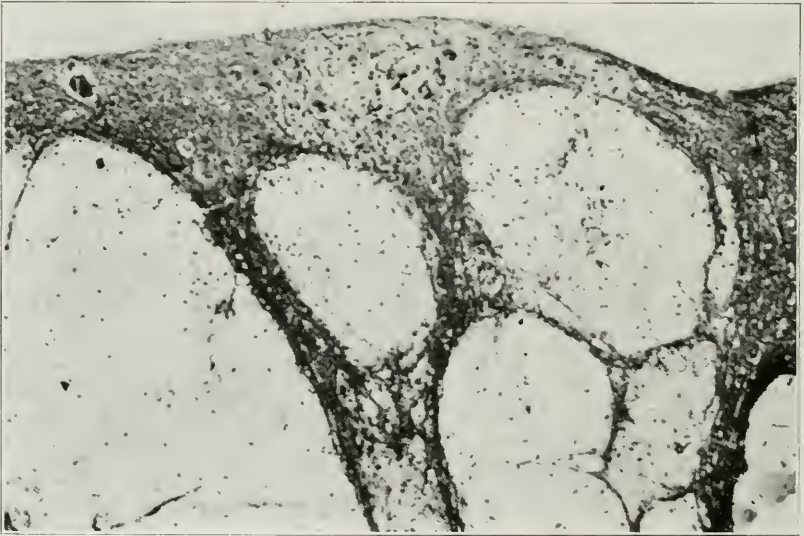


Fig. 2.—Part of a multilocular cyst, close to the surface. The most superficial cyst is recent. The others have enlarged as the result of exudation and expansion of the mucoid envelopes of the blastomyces.



Fig. 3.—Phagocytic giant cells in cavities. A few organisms are free in the cavity. The insert shows a more highly magnified giant cell. Frozen section. Toluidin blue.

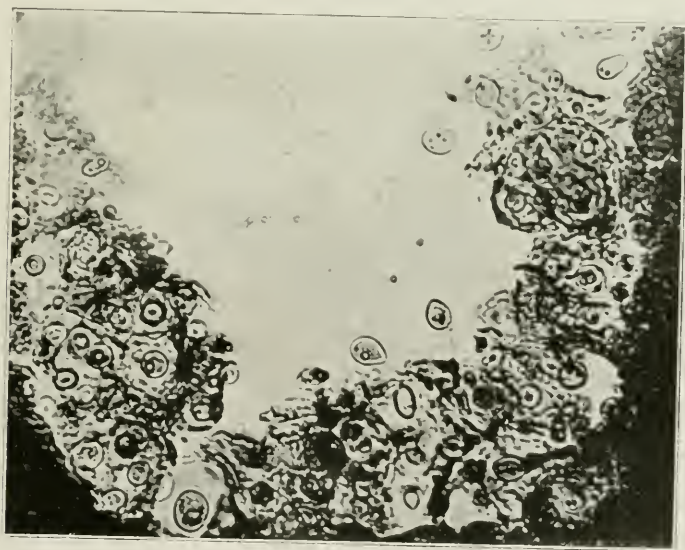


Fig. 4.—Torulae in a cyst. The morphology of the organisms is well shown. The halos are vague. On the right is a multinucleated phagocytic cell. Frozen section. Toluidin blue.

had been observed during life. There was a focus also in the pyramidal tract where the myelin sheaths were disrupted and a cystic condition was developing. The optic nerve contained numerous small cysts of spindle shape, which lay between the fasciculi and separated them. The myelin sheaths were not invaded and did not appear swollen or otherwise degenerated. The meninges over the base were moderately infiltrated with fluid, yeast cells, and phagocytes. Many of the vessels had large collars of these cells (Fig. 6).

The spinal meninges were more compact than the cerebral, and here and there blastomyces were found. Giant cells were scarce. The cord substance was not invaded by the organisms. There was no degeneration in the projection tracts.

Histologic Summary.—The cerebral cortex, basal ganglions and cerebellum showed great numbers of minute cysts which varied markedly in size. They contained blastomyces, phagocytes, fluid and detritus. Destruction in both gray and white matter seemed to have resulted from the pressure due to the growth of the cysts. The reaction on the part of the host's tissues was almost



Fig. 5.—Torula with a capsule. Frozen section. Basic fuchsin.

negligible. There was no neuroglia proliferation, and little or no inflammatory cell exudate. The discovery of yeast cells in the dilated perivascular sheaths suggests that extension followed this route.

THE FUNGUS

Technic.—In tissue which has been embedded, many yeast cells, especially the young forms, suffer so much distortion that they may be unrecognizable as yeasts. Even with the mature forms, it is better to avoid embedding processes and to use frozen sections to bring out the finer structural details, and to examine the tissue either when unstained or after staining with watery solutions of toluidin blue, thionin or basic fuchsin. The yeast cells should be examined in a watery or alcoholic medium. Clearing the sections with glycerin or xylol is to be avoided. To bring out the mucinous envelope, we stained over night at 50 C. with dilute aqueous solution of basic fuchsin. In such tissue preparations, budding and younger forms of the yeast were scarce, and the coarsely granular forms with double contour were numerous. The extracellular yeast

cells tended to be small, to contain small numbers of granules, to be sometimes apiculate or ellipsoidal, and frequently to have double contours. Their form thus corresponded to that of old cultures (adverse environment). Intracellular forms were larger, had larger and fewer granules, thick shells, and sometimes a very heavy hyaloid (mucinous) coating. This mucinous envelope did not give the tinctorial reactions of mucin and was eventually demonstrated in material from cultures as well as in the tissues.

Cultural Characters.—Primary cultures were obtained from spinal puncture fluid only on blood agar. Trials on plain mediums were unsuccessful but subcultures thereafter grew well. These primary cultures, and the earlier ones on potato, lacked the strong yellow color of later subcultures. On blood agar

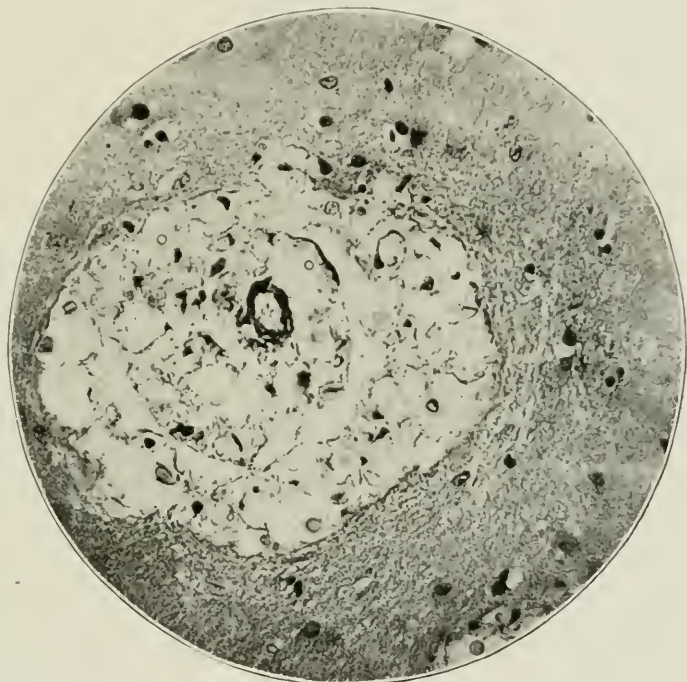


Fig. 6.—Extension of yeast cells along a perivascular space. Yeast cells, phagocytes and detritus are shown. Hematoxylin-eosin.

they were white, and on potato yellow-gray. On the latter the growth was very abundant. Further observations were made over a period of several months.

The organism grew well at body temperature, and equally well at room temperature in semi-darkness, on glucose agar, plain peptone agar, beer-wort, and glucose agar to which gentian violet, in the proportion of 1:100,000 had been added. Plantings on solid mediums included both plate and tube plantings. As usual with yeasts, growth was most luxuriant on the several carbohydrate mediums. The growth on glucose agar will be described as the type, and the variations on the other mediums will be noted later.

In tube cultures, the outstanding naked eye characters of the colonies were their rapidity and luxuriance of growth, the strong yellow color, the uniformly

round contour and the highly glistening surface. As compared with control cultures—*Saccharomyces cerevisiae*—this yeast grew fully as rapidly at first, and ultimately excelled and developed even to twice the bulk of the control. The margin of the colony was sharp, moderately elevated, rounded, and regular. The yellow color developed only after four weeks in the earlier cultures, but in later subcultures it developed much more promptly, in from seven to ten days. The organism did not penetrate downward into the medium nor discolor it in the least. When the tube cultures were from six to eight weeks old, or when mediums for any reason began to dry out, the colony reflected the desiccating process; its color became yellowish-brown, and its surface less glistening. But it never developed any special surface markings such as furrows, wrinkles, foldings, peripheral radiations, or wedges. On no kind of medium, solid or liquid, was there ever any naked-eye evidence that mycelia were being produced.

On gentian-violet glucose agar, the characters of the colony itself were unchanged. The organism was very resistant to the dye, and it was necessary to add gentian-violet to the medium in the proportion of 1:1,000 before growth was restrained. This property may be useful in securing pure primary cultures from tissues.

On plain peptone agar (containing no carbohydrates) a non-characteristic white colony, three to four millimeters in diameter, developed. By the naked eye it could not be distinguished, at any age, from many other yeasts. It had the same general, more or less dirty-white, homogeneous, pasty character.

In beer-wort, Guilliermond's formula,¹ the growth became evident by a faint cloudiness of the medium, and a delicate ring of scum which was attached to the walls of the tube at the surface of the medium. By the end of two weeks, the scum had become a thin pellicle; there was a granular growth adherent to the sides of the tube from the top to the bottom, and a moderately heavy sediment. The medium was clear until agitation dislodged the surface particles.

When grown in Petri dishes, the changes in color and consistency proceeded much more rapidly than on the thicker strata of medium in tubes; and the colonies, even when the medium was prevented from drying out, never grew as large. The streaks had the common pasty, homogeneous character of yeasts. They showed a yellow color as early as the fourth day and in four weeks they became brownish-yellow. The streaks never become more than a centimeter wide; the edges never were fuzzy enough to indicate mycelial development; and none of the lumpy characters, such as Türk has described for his yeast, were ever developed.

In glucose-agar stabs at the end of twenty-six days, the surface of the culture was completely covered by a colony of burnt-orange color. In the depths of the stab there was a well developed growth, almost as heavy at a depth of five centimeters as at the surface. Fine granules could be seen projecting from the margin of the stab. There was no evidence of gas production.

In fermentation tests it was found that late subcultures of the organism split sugars slowly. No gas was produced in glucose, maltose, saccharose, lactose, galactose, inulin or dextrin mediums at any time. Acid was produced in only four of these seven sugars (saccharose, glucose, maltose and dextrin), and then but slowly.

1. Guilliermond, A.: The Yeasts, English translation by Tanner, New York, John Wiley & Sons, 1920, p. 150.

Morphology.—Our yeast was identical morphologically with that of Türk and, since he has so well portrayed it, we shall but briefly describe it. The appearance of individual yeast cells varied markedly in accordance with their age. Mature forms were spherical with double contour, pale green in color, and of about the same size as *Saccharomyces cerevisiae*. The earliest budding examples were coarsely granular, but in a few days cultures were found also to contain large cells with a double contour and a solid, hyaloid, spheroid body, which almost or quite filled the interior. In cultures several weeks old the central part of such a cell contained, instead of the single body, from two to eight or more larger or smaller hyaloid spheroids. These were variable both in number and size. We, therefore, finally concluded that they were not asci. Buds could be found springing from any of the above forms of cell after three weeks, in all the mediums used, solid or liquid.

Inasmuch as the subject of asci is so important for classification, we tried all of the cultural and staining methods which have been recommended by

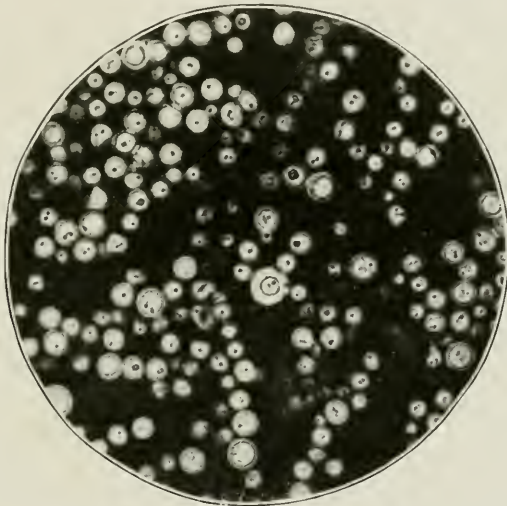


Fig. 7.—Cells from two months' old culture showing mucoid capsules. India ink method.

Guilliermond for bringing out these structures, and controlled our results with *Saccharomyces cerevisiae*. Asci failed to appear and we finally concluded that this organism does not produce them.

Mycelial structures were found in only four of the culture tubes from a dozen examined, and then in the condensation-water at the bottom of the glucose agar tubes which were two months old. Hence, it must be concluded that the filamentous form of this organism is most exceptional. It is not to be considered in classification.

Attempts, by staining methods, to demonstrate a mucinous envelope on organisms from culture were unsuccessful. We finally detected it by making an emulsion of yeast cells with India ink and examining them at once under a coverslip (Fig. 7). A brilliant, refractile halo appeared around many of the older cells, but only from cultures several months old. The substance of this envelope must be a product of old cells only, and probably of cells that are experiencing an unfavorable environment.

CLASSIFICATION OF FUNGUS

In the present stage of yeast classification, there is but little incentive to attempt to place pathogenic yeasts into exact taxonomic positions. Guilliermond¹ following Vuillemin, groups all the pathogens which do not sporulate into the single genus, *Cryptococcus*. Castellani² in his Milroy lectures has outlined a classification on a strictly morphologic basis. By that also this cerebrospinal blastomyces falls into the genus *Cryptococcus*. The absence of ascus production eliminates both the saccharomycetes and endomycetes from consideration. *Monilia* exhibits mycelial forms much more constantly than does our cerebrospinal specimen, and also develops chains of cells; it is therefore easily excluded. *Torula* and *mycoderma* are characterized, among other things, by the development of a slimy scum in culture which was never present in fluid cultures of our organism.

Guilliermond describes no species which coincides exactly with our specimen. It is true that his work was mostly concerned with such industrial yeasts as interest the brewer and baker; yet there is a fairly full section devoted to pathogenic forms. The medical literature contains more or less full descriptions of blastomyces which are parasitic in the human cerebrospinal axis, and these require the most careful comparison with ours. For the most part, however, the cultural reports are incomplete or inconclusive. Five samples of blastomyces from the central nervous system have been described with sufficient detail to justify analysis.

Swift and Bull's Case.—The organism grown by these authors appeared and behaved similarly to ours. Primary cultures were obtained only on Loeffler's blood serum and developed slowly; subcultures grew freely and became yellow with age. No fermentation tests were recorded. They described an encapsulated form which was seen only in the spinal fluid, and not in cultures or sections. The capsule was certainly inconspicuous and was not specifically investigated. A second important difference was in the matter of mycelia. We saw them in one culture tube only, and that was a subculture; Swift and Bull found them only in primary culture. Otherwise, the microscopic appearance tallied with that of our organism. Their organism, however, was not intracellular and did not form cysts.

Benda's Yeast.—The cultural data given were very limited and concerned almost exclusively the acid requirements. Glucose was never split. It was an obligate aerobe, and was unable to grow at the bottom of bouillon tubes. Morphologically, the author describes the yeast only in tissue. He does not mention ascus production nor a mucinous envelope.

2. Castellani, Aldo: J. Trop. Med. **23**:101 (May) 1920.

Türk's Yeast.—The characters of this yeast were most minutely described. Microscopically the cells were identical in appearance with ours, and this is well brought out in excellent illustrations. In culture likewise, the two yeasts behaved alike; they grew freely and became yellow. For our purpose, the differences are more useful.

They are: (1) Türk's yeast, unlike ours, developed peripheral translucent wedges (Zwickel). Our variety was grown in company with thirteen other species, during a period of over a year. We made numerous subcultures on different mediums. Other species developed the wedges but they never appeared in the tubes or plates of our yeast. (2) Our yeast frequently became deep orange-yellow in color.

Flu and Wocnsdreght's Yeast.—This organism had the same characters as ours both microscopically and culturally, except that the primary cultures grow on Sabouraud's medium. These authors report that their organism developed no spores (or asci) in special tests. There was no fermentation of glucose, saccharose or mannite.

Williams' Yeast.—This author speaks of the lesions in his case as abscesses but he does not mention that the fluid was purulent, nor that leukocytes featured in the histologic sections. We presume, therefore, that the lesions were really cysts. Cultural studies did not include the use of solid sugar mediums, so that there was no opportunity for the yellow color to develop; but a leaning in that direction is indicated by the yellowish white color of plain agar cultures. This organism appears to have been identical with ours except that growth of his cultures was inhibited at room temperature. Williams emphasizes the stickiness of the growth and observed mycelia in the kidney of an inoculated rabbit.

Frothingham's Yeast.—Stoddard and Cutler did not secure cultures from their two patients, but they restudied Frothingham's material from a horse, and their very full description leaves no doubt that our organism was the same as Frothingham's. The only points that we wish to restate are that no sliminess or pellicles developed in their fluid mediums, and that they mention no intracellular fat bodies; both of these are usual in the *Torula* in which they place their organism. The mucinous envelope around the cells was observed in culture, and unlike ours, became stained with methylene blue, Wright's, and Giemsa's stain.

These same authors are responsible for the name "*Torula histolytica*" which we have accepted for our yeast; they applied it, we assume, both to the organisms of the human cases, and to Frothingham's horse cultures. In placing this yeast in the genus *Torula*, they devote no special paragraph to the diagnosis of the established genus

Torula Turpin and its comparison with the characters of their yeast. Clearly, their comparative table is directed at the three classes of yeast infection that they studied and not at any taxonomic divisions of the microorganism. We take it therefore, from sentences scattered through the text, that they have cut the Gordian knot and have placed the yeast in the *Torulac*, more on the basis of the type of disease produced (influenced by Wollbach), than in conformity to the usual procedures of the general biologist. On the basis of the latter we feel that the name *Cryptococcus histolytica* would be more correct; nevertheless we accept the name *Torula histolytica* for our organism, not so much on the ground of its appropriateness as to avoid adding to the confusion of a frontier subject like yeast classification.

After reviewing the seven yeasts that have been cultured (including the one here reported), the question arises as to which were *Torula histolytica*. Two are so fully described as to permit us to say that this organism can be incriminated in Frothingham's and Flu and Woensdreght's cases as well as our own.³ The cases of Stoddard and Cutler are not acceptable as they did not grow the organisms; an identical appearance of the yeast in the tissue and identical effect on the tissue do not guarantee that the same microorganism is at work. Türk's data are so complete as to differentiate his organism from *Torula histolytica*. The remaining cases of Swift and Bull, Benda, and Williams are doubtful. But there can be no doubt that several, at least, of these and the cases without cultures were caused by *Torula histolytica*.

ANIMAL INOCULATION EXPERIMENTS

When guinea-pigs were inoculated intraperitoneally with spinal fluid or fresh cultures they lost weight rapidly and died in from four days to two weeks, without specific alterations in the brain or other organs. As a rule we were unsuccessful in recovering the organisms by culture or smears, and none were found in the sections. Inoculated intranasally, the period of infection lasted from two to three weeks and at necropsy the organisms were demonstrable by smear and culture from various organs, and by histologic examination in the cerebral meninges.

The only successful reproduction of the cystic disease took place in a guinea-pig which was injected subcutaneously. Six months later the animal was found dead in its cage. The testes were greatly enlarged and broken down; and sticky, yellow pus was being discharged from several sinuses. Grossly, the internal organs appeared normal and were not examined histologically. No abnormality was noted in the fresh

3. Frothingham's yeast was obtained from the lung of a horse; there are, therefore, only two cystic brain cases for which the responsibility of this organ is established.

brain but, on histologic examination, numerous multilocular cysts containing yeast cells were found, which corresponded almost exactly with the condition in the human brain. In the guinea-pig there was a moderate, perivascular, round-cell infiltration, which the human case did not show, but the formation of giant cells was similar. Examination of the testes revealed invasion of the gland here and there by organisms; in the subcutaneous tissue there were multilocular cysts of large size, which gave the whole area a reticulated appearance. In these cysts there were yeast cells, both free and in phagocytes, just as in the brain. Flu and Woensdrecht, Evans,⁴ and Stoddard and Cutler⁵ reported similar results, and Klarfeld⁶ had produced cystic infiltration of the cerebral gray matter in dogs with a different organism.

FREQUENCY

Cystic infiltration of the gray matter similar to that described here has been recorded by Zenker,⁷ von Hansemann,⁸ Benda,⁹ Flu and Woensdrecht,¹⁰ Riley and Le Count,¹¹ Rusk and Farnell¹² (two cases), Williams,¹³ Stoddard and Cutler,⁵ and Pierson.¹⁴ All of these authors emphasized the irregular distribution of the cysts, their limitation to the gray matter and the absence of tissue reaction. Giant cells are mentioned in several reports, although Rusk and Farnell believed them to be mere zooglear masses of the organisms. Other authors have

4. Evans, Newton: *Torula Infection*, California State J. of Med. **20**:383, 1922.

5. Stoddard, J. L., and Cutler, E. C.: *Torula Infection in Man*, Monograph No. 6 from the Rockefeller Institute for Medical Research, New York (Jan. 31) 1916.

6. Klarfeld: *Zur Histopathologie der experimentalen Blastomykose des Gehirns*, Ztschr. f. d. ges. Neurol. u. Psychiat. **58**:176, 1920.

7. Zenker: *Encephalitis mit Pilzentwicklung im Gehirn*, Jahres. d. Gesellsch. f. Natur. u. Heilk. in Dresden, 1861-1862, p. 51.

8. Von Hansemann: *Ueber einen Fall von Hefenerkrankung*, Centralbl. f. allg. Path. u. path. Anat. **16**:347 (July 1) 1905.

9. Benda: *Ein Fall von Blastomykosis Cerebri*, Deutsch. med. Wchnschr. **33**:945, 1907.

10. Flu and Woensdrecht: *Een Geval Blastomycose van het Centraal-zenuwustelsel*, Meded. v. d. burgerl. Bencesk. dienst. in Nederl. Indie **6**:1, 1918.

11. Riley, F. B., and LeCount, E. R.: *A Case of Systemic Blastomycosis*, Arch. Int. Med. **13**:614 (April) 1914.

12. Rusk and Farnell: *Systemic Oidiomycosis. A Study of Two Cases Developing Terminal Oidiomycotic Meningitis*, Univ. California Pub. Pathology **2**:47, 1912.

13. Williams, J. R.: *A Case of Systemic Blastomycosis*, M. J. Australia **2**:185, 1922.

14. Pierson, P. H.: *Torula in Man*, J. A. M. A. **69**:279 (Dec. 29) 1917.

described lymphocytes and plasma cells about the cysts and in the meninges, and Zenker and Williams described the cysts as small abscesses.

It is a question whether the cysts are formed by internal expansion of the proliferating organisms, or by lysis of tissue due to the specific biologic activity of the organisms. Stoddard and Cutler believed the latter and named the organism *Torula histolytica* on account of this supposed action. We are not satisfied that this is entirely the case. In the first place the surrounding tissue shows evidences of compression. In the second place the septa are formed by well preserved nervous tissue; thirdly there is not enough fatty material in the cysts and their vicinity and about the vessels to point to active destruction; and lastly the organism when grown on fresh sterile brain, produced no solution of the medium.

Cases of meningitis without cyst formation have been reported by Türk,¹⁵ Swift and Bull,¹⁶ Versé,¹⁷ Goto,¹⁸ and Tedesko,¹⁹ in the case of Curtis²⁰ and the two cases reported by Evans no necropsy was made. These cases are included in order that diffuse blastomycosis of the central nervous system may be better understood.

In these twenty cases (which include that here reported) of diffuse meningeal blastomycosis no extracranial lesion was found in five cases. In the cases of Zenker, Flu and Woensdreght, and Türk, exudates containing blastomyces were present in the pharynx. In Stoddard and Cutler's Case 1, there was an exudate in the pharynx that was not examined. Five patients presented lesions in many organs, and five had pulmonary involvement.

PORTALS OF ENTRY

The cases of predominant or "primary" blastomycosis of the central nervous system are of special interest on account of the question of the portal of entry. The pharyngeal exudates in the cases of Zenker, Türk, and Flu and Woensdreght may have been terminal, for they appeared shortly before death. Moreover, Flu and Woensdreght, after intracardiac injection of the organism, recovered it from the nasal cavity as well as from the brain. The lung is certainly one portal of entry; not only are five cases reported in which there was coincident

15. Türk: Ein Fall von Hefeinfektion (Saccharomykose) der Meningen, Deutsch. Arch. f. klin. Med. **90**:367, 1907.

16. Swift and Bull: Blastomycotic Meningitis, M. J. Australia, **2**:265, 1917.

17. Versé: Ueber einen Fall von generalisierten Blastomykose beim Menschen, Verhandl. d. deutsch. path. Gesellsch. **17**:275, 1914.

18. Goto: Ueber Blastomycetenmeningitis, Mitt. a. d. med. Fak. d. k. Univ. Tokyo **15**:75, 1915.

19. Tedesko, quoted by Helly in Discussion on Versé (Footnote 13).

20. Curtis: Presse méd., 1895, p. 370.

pulmonary involvement, in addition to five with generalized blastomycosis, but in three cases ulcerative tuberculosis was found, in one bronchiectasis, and in another residual influenza. Thus in fifteen of the twenty cases, the lungs were diseased. The alimentary tract forms another possible portal of entry. Tanaka²¹ recently reported the isolation of several species of blastomyces from the mesenteric nodes of persons who had died from various diseases. We obtained our organisms from a peripancreatic lymph node. In our case the portal of entry may have been the tonsils, for the cervical lymph nodes became enlarged after tonsillectomy, and one of these nodes, when excised five years later, presented appearances not unlike those found in the axillary and mesenteric lymph nodes at necropsy.

SUMMARY

1. We report a twelfth case of cystic infiltration of the cerebral gray matter with a blastomyces.
2. The clinical picture is that of tuberculous meningitis or unlocalized brain tumor, but the organisms are readily found in the spinal fluid.
3. The organism in our case was *Torula histolytica* Stoddard and Cutler.²²
4. The route by which the brain becomes infected is in doubt.
5. The organism excites hyperplasia of the meninges and the formation of phagocytic giant cells. It provokes no reaction either of neuroglia or connective tissue elements in the cerebral substance, and causes no infiltration with leukocytes. It forms cysts in the cerebral gray matter by internal expansion and exudation. It probably travels along the perivascular lymph spaces.
6. The disease was transmitted to guinea-pigs. These developed a general infection, and, in one animal, there was cystic infiltration of the brain and testes.

21. Tanaka: *Saccharomyces in Mesenteric Glands of the Human Body*, J. Path. & Bacteriol. **23**:350, 1920.

22. Cultures of *Torula histolytica* have been deposited in the National Collection for Type Cultures, Lister Institute, Chelsea Gardens, London. Culture and histologic sections have been deposited at the Army Medical Museum, Washington, D. C.

EXPERIMENTAL CONVULSIONS

A CONSIDERATION OF THE EPILEPTOGENOUS ZONES OF THE
CENTRAL NERVOUS SYSTEM *

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Since the discovery by Fritsch and Hitzig¹ of the electrical irritability of the brain, numerous theories concerning the site of convulsive discharge have been proposed. It is unfortunate that the site of the convulsive discharge has been considered inseparable from the pathology of the disease, epilepsy. It is further unfortunate that much of the work has been concerned with the establishment of a so-called convulsive center. These conditions are in part responsible for the existing confusion relating to the pathogenesis of epilepsy.

Kussmaul and Tenner² in 1857, succeeded in producing convulsions in rabbits as the result of cerebral anemia. They were further able to evoke such convulsions after removal of the cerebrum, and pointed out the importance of the brain stem in the production of the convulsion. Nothnagel³ then localized a special convulsive center in the pons and medulla. It was pointed out by Bubnoff and Heidenhain⁴ that both cortical and subcortical lesions are concerned in the production of a convulsion. Upon experimental grounds Ziehen⁵ proposed that the clonic component of the convulsion was purely cortical, whereas the tonic component was of subcortical origin. He showed that, when, in the presence of a tonic-clonic convulsion, the cortex was extirpated, the clonic element disappeared. It was possible for Binswanger⁶ to produce convulsive attacks by irritation, electrical or mechanical, of the pons, but the attacks were tonic in nature.

The production of convulsive seizures by electrical irritation of the cortex then brought the cortical theory of convulsions to the foreground.

It is apparent therefore, that there are three chief theories as to the location of the convulsive site: First, that which deals with the cortex alone (Hitzig and Fritsch, Luciani, Unverricht, and others);

* From the Nelson Morris Memorial Institute for Medical Research of the Michael Reese Hospital and Northwestern University Medical School.

* Read at the Third Annual Meeting of the Association for Research in Nervous and Mental Diseases, Dec. 28, 1922.

1. Fritsch and Hitzig: *Archiv. f. Anat. u. Physiol.* p. 300. No. 2, 1870.

2. Kussmaul and Tenner: London, New Sydenham Society, p. 105, 1859.

3. Nothnagel: *Virchow's Archiv.* **44**:1, 1868.

4. Bubnoff and Heidenhain, R. P.: *Pflüger's Archiv.* **26**:137, 1881.

5. Ziehen, Theodore: *Archiv. f. Psychiat.* **17**:99, 1886.

6. Binswanger: *Archiv. f. Psychiat.* **19**:759, 1887.

second, the combined cortical and subcortical theory (Ziehen, Bubnoff and Heidenhain, Binswanger, Hughlings Jackson, and others); and, third, the medullary theory (Kussmaul and Tenner, Van der Kolk, Nothnagel, and others).

The experiments in support of these theories have been of two types: first, those in which convulsive seizures have been produced by irritation (mechanical, chemical or electrical) of various parts of the central nervous system; and, second, those in which experimentally produced convulsions have been modified or stopped by ablation of certain parts of the central nervous system, or their occurrence has been prevented by such ablation (excision, separation, freezing, and cocainization).

The proponents of the medullary theory have assumed that if convulsions may be produced by irritation of the pons and the medulla, and may occur as the result of intoxication with absinthe, etc., in decerebrated animals, the convulsive site is located in the pons and medulla, or the pons and medulla possess epileptogenous properties. Opposed to this are the observations: that when a convulsion occurs as the result of irritation of the pons, the clonic element disappears after the removal of the cerebrum (Ziehen, Bechterew, Lewandowsky); that when the cerebral hemispheres are extirpated, it is impossible to produce a convulsion possessing tonic and clonic elements; that when convulsive seizures are produced by toxins, such as absinthe, the clonic element of the attack disappears after the removal of the motor cortex (Borischpolsky and Ossipow); and that removal of one cerebral hemisphere causes the convulsions to disappear from one-half of the body. The observations relative to toxic convulsions are not unchallenged, as Albertoni⁷ was able to produce in dogs, after full recovery from a section below the optic thalami, a generalized bilateral convulsion by the use of cinchonidin. Grünwald⁸ observed typical picrotoxin convulsions in a decerebrate cat and dog, and subsequent workers have made similar observations.

The proponents of the mixed theory are dependent upon observations of certain motor manifestations produced by stimulation of various parts of the central nervous system. Many of these experiments have nothing to do with convulsions. They are concerned with the production of certain forced movements, ataxia and dystonia; included among them are the experiments of Flourens, Schiff, and Majendie on the quadrigeminal bodies, of Nothnagel, Albertoni, Michieli, Lussana and others on the basal ganglions.

7. Albertoni: *Archiv. f. exper. Path. u. Pharmakol.* **15**:258, 1882.

8. Grünwald: *Archiv. f. exper. Path. u. Pharmakol.* **60**:249, 1909.

The evidence which has supported the theory of the cortical origin of convulsive seizures has been supplied chiefly by the observation of convulsions produced by electrical stimulation of the cortex. Here it has been found that convulsions may be produced which resemble epilepsy, (Fritsch and Hitzig, Unverricht, Ferrier and others). It has been shown that if electrical stimulation of the motor cortex is long continued, the resulting isolated contractions of muscle groups become successive and spread to muscle groups innervated by adjacent cortical areas; then they spread to the opposite side, involving, usually, the posterior extremity first, then the anterior extremity and then the face, although variations occur. The resulting convulsion outlasts the electrical stimulation. It has been shown: that if one quickly extirpates an irritated zone the convulsion is inhibited; that after deep decortication, such convulsions cannot be produced; that when certain motor areas have been removed, the movements represented by such areas are absent in an ensuing convulsion; and that the spread of a convulsion may be stopped by deep incisions into adjacent areas of the cortex. Convulsions can be produced not only by irritation of the motor cortex but also by irritation of other cortical areas, for example the occipital region.

It may be assumed to be essential that the cerebral cortex be intact for the production of a cortical fit. Granting that convulsions may be produced by irritation of the cortex, can they be produced by irritation of the subcortical regions? It has been contended that pontine hemorrhage has never produced convulsions and that spontaneous convulsions never occur in decerebrate animals. Luce⁹ has observed the occurrence of convulsions in pontine hemorrhage and stated that such convulsions are produced by irritation of the motor field of the pons, because they occurred in his case when connections with the cerebral cortex were destroyed by hemorrhage. He believes that the ganglion cells of the pons possess epileptogenous qualities, and that in man it may be proved that, in addition to cortical epilepsy, a subcortical type exists. He stated, however, that, in his opinion, the pons plays only a secondary part in genuine epilepsy. Rothmann¹⁰ has reported the occurrence of spontaneous convulsions in a decerebrated dog, three months after the operation. The convulsive seizure was tonic in the extremities and clonic in the face. This observation likewise points to the epileptogenous quality of the brain stem. From these observations and from the accumulated experimental work it may, I think, be admitted that, whatever element of a fit may be lacking in a convulsion produced in an animal with a mutilated brain, convulsions, tonic,

9. Luce: *Deutsch. Zeitschr. f. Nervenheilk.* **15**:327, 1899.

10. Rothmann: *Neurol. Centrbl.* **31**:1286, 1912.

clonic, or both, may be produced from the cortex and from the sub-cortical regions; and in the latter case, in the absence of the motor cortex. It may further be admitted that the symptoms of a convulsion vary with the state of preservation of the brain; if it be intact there are: tonic-clonic convulsions associated with sialorrhea, pupillary changes, bladder contraction, circulatory and respiratory changes, and at times an aura.

The aura is dependent on the area of brain irritated, and in Jacksonian epilepsy, as in artificially produced cortical fits, motor movements constitute the aura. In other cases vagal auras exist, or respiratory or circulatory changes precede the convulsion; the portion of the brain here irritated must be at a lower level. It may be pointed out that not only is the tonic element of an epileptic fit absent in a cortical fit, but that loss of consciousness does not occur until a generalized convulsion ensues; involuntary urination does not occur before the loss of consciousness.

An epileptic convulsion consists not only of tonic and clonic movements, but also of a series of events and changes that include the aura, vascular, respiratory and secretory changes, and contraction of the involuntary muscles of the bladder. It has been shown that by irritation of certain parts of the cortex many of these changes may be produced: pupillary changes (Lewandowsky), bladder contraction (Bechterew), circulatory and respiratory changes (Landois), and secretory changes of the salivary glands (Kulz and Braun). These regions are rather widely separated from each other and if it could be shown that all of these changes can be evoked from some relatively small point, their occurrence in epilepsy might be more readily explained. It has been pointed out by Hirt that such symptoms occurring in an epileptic attack have never been adequately explained.

Certain toxic convulsions have thrown some light on this question. The occurrence of convulsions produced by toxic substances in decerebrate animals has been described by Roeber,¹¹ Perrier,¹² Grünwald and others, who worked with picrotoxin; Albertoni with cinchonidin; Ossipow with absinthe; and others with various substances such as toxiresin, digitaliresin, coriamyrtin, senso, etc. Not only do these substances produce convulsions which, whatever be their pattern, occur after decerebration, but, as was pointed out by Grünwald,¹³ they also produce a marked effect on the central autonomic nervous system. He remarked that a review of the literature showed that, in addition to the production of convulsions and injury to the heart muscle, picrotoxin caused: contraction of the pupils (Falck, Roeber, Luchsinger, Perrier):

11. Roeber: *Archiv. f. Anat. u. Physiol.* p. 38, No. 1, 1869.

12. Perrier: *Archiv. f. exper. Path. u. Pharmacol.* **4**:191, 1875

13. Grünwald: *Archiv. f. exper. Path. u. Pharmacol.* **15**:258, 1882.

salivation (Falck, Roeber, Luchsinger, Gottlieb); slow pulse (Roeber, Perrier, Luchsinger, Gottlieb); and urinary bladder contraction (Falck and Gottlieb). He verified these findings and demonstrated the central action of the poison as follows: after cutting the bladder nerves no contraction occurred following the administration of picrotoxin. In a decerebrate cat that had been poisoned with picrotoxin and showed a vagus pulse, salivation and bladder contraction, these three symptoms disappeared on section of the vagus, chorda tympani and pelvic nerves respectively; the symptoms did not recur on further administration of picrotoxin. After section of the right chorda tympani, following the administration of picrotoxin, there was profuse secretion from the left salivary duct but none from the right. From these experiments he concluded that picrotoxin is a poison that acts on the central autonomic nervous system. It can readily be seen that picrotoxin may produce convulsions through action on the medulla and at the same time produce a group of symptoms referable to the central autonomic nervous system, which regularly occur in conjunction with an epileptic convulsion.

With W. L. Treadway¹⁴ I pointed out that certain circulatory and respiratory changes occurred in relation with an epileptic convulsion. We found that in a case of petit mal remarkably well suited for continuous tracings, there was a preliminary rise in blood pressure twenty-six to sixty seconds before the convulsion; when the rise took place more than thirty seconds before a convulsion, the blood pressure usually fell again to a slight degree. Preceding the convulsion by from nine to twelve seconds, there was a sudden marked drop in blood pressure which remained relatively low during the time occupied by the petit mal attack. The aura followed the blood pressure change. A period of apnea preceded the convulsion or its equivalent by from four to nine seconds.

Knauer¹⁵ has observed inhibitory spasm of respiration in the beginning of an epileptic attack which he attributed to spasmodic closure of the glottis. In reviewing the work of Treadway and myself, I was astonished that the obvious fact that apnea occurred in inspiration escaped our notice. The apnea certainly was not due to closure of the glottis, which would not interfere with movements of the chest, yet this observation makes it necessary to show that the apnea is not the result of the tonic element of the fit. I have been fortunate in obtaining a series of tracings of the respiration and blood pressure of a girl in status epilepticus. In these it can be seen that, preceding the convulsion, there was a cessation of respiration in the expiratory phase

14. Pollock, L. J., and Treadway, W. L.: *Arch. Int. Med.* **12**:445, 1913.

15. Knauer: *Zeitschr. f. d. ges. Neurol. u. Psychiat.* **3**:625, 1910.

with a few very ill-defined movements of the chest wall and then an inspiratory spasm. Unfortunately, the blood pressure curve was valueless because of a defect in the Erlanger valve. It may be well to point out that in picrotoxin convulsions the apnea sometimes occurred at a point midway between inspiration and expiration and usually in expiration. Although Grünwald attempted to show that the blood pressure changes were due to respiratory embarrassment, we have pointed out that the rise and fall of blood pressure at times occurred independently of the period of apnea. At times respiration may only be diminished, no period of apnea being present; and at other times respiration may actually be increased, and blood pressure variations may occur. I feel, therefore, that the circulatory and respiratory changes occur as the result of medullary stimulation and not as the result of muscular movement or respiratory embarrassment.

From the facts that respiratory and circulatory disturbances preceded the convulsion or equivalent, the conclusion was reached that in some cases of petit mal the site of discharge is in the medulla and pons.

If convulsions which are caused by discharge in the medulla and pons are accompanied by a train of circulatory and respiratory symptoms, it would seem likely that these symptoms should be present in a medullary convulsion artificially produced.

W. H. Holmes and I¹⁶ studied respiration and circulation in picrotoxin convulsions and found that a period of apnea occurred with the convulsion; it usually began with the severe clonic seizure, but sometimes followed it. We likewise found that there was a periodic rise and fall in blood pressure as part of the manifestation of picrotoxin poisoning; this took place about the time of the convulsion but without having a definite relation to it, or being caused by it or the period of apnea. Similar circulatory changes occurred in a decerebrate animal. It was found that although apnea and blood pressure changes occurred with the convulsion caused by picrotoxin, they did not bear the same constant relation to the convulsion as was found in the case of petit mal. From the facts that there exist medullary convulsants and that the convulsions which they produce are associated with symptoms referable to the central autonomic nervous system similar to those found in idiopathic epilepsy, and with circulatory disturbances analogous to those found in idiopathic epilepsy, we concluded that we had further evidence that the site of discharge of the convulsion in some types of idiopathic epilepsy is in the medulla and pons. The medulla and pons participate in the convulsion in all cases of epilepsy, whether the site of discharge is there or not.

16. Pollock, L. J., and Holmes, W. H.: *Arch. Int. Med.* **16**:213, 1915.

It has become necessary to define what is meant by "site of discharge." By this term is meant that area of the central nervous system which, because of stimulation of a certain character, evokes the beginning of a convulsion. It does not signify a special center for convulsions, but only that such an area possesses epileptogenous quality. With this in mind, it appears to me just as logical to assume in those cases in which the first sign of convulsion may be referred to the central autonomic nervous system, that the irritation producing the convulsion is first expended upon the brain stem, as it is to attribute the origin of Jacksonian fits to the cortex. The objection of the proponents of the cortical theory of epilepsy, that in subcortical fits the clonic element is absent, would, even if it were true, be equivalent to the objection that in a cortical fit the tonic element, which is always observed *first* in an epileptic convulsion, is absent.

In the present work which, unfortunately, is limited, it was proposed to determine whether, in picrotoxin poisoning, an isolated segment of the brain stem could evoke convulsive seizures in the muscles innervated by this segment.

Twenty-eight animals, eight cats and twenty dogs, were studied, and a large number of frogs were observed under certain conditions. Of the cats and dogs, ten animals afforded successful experiments. Of these, three were decerebrated by a transection of the brain stem below the optic thalami; four by transection immediately below the level of the nucleus of the seventh cranial nerve; and three were transected at two levels, one below the optic thalami and the other immediately above the level of the nucleus of the seventh cranial nerve. Of the last group, one received a hypodermic injection of picrotoxin solution following the section in the pons above the seventh nerve. The other two received the injection following the double transection.

The section above and below the level of the seventh cranial nerve was performed by two methods, the first having an approach through a cerebellar decompression opening, and the other through the sphenoid bone in the roof of the mouth. A satisfactory technic was developed by both methods. Intratracheal artificial respiration was employed and ether used as an anesthetic. The dose of picrotoxin employed was about eight milligrams per kilogram of body weight of the animal.

As had been observed before, it was readily seen that a number of factors were responsible for differences in the pattern of convulsive movements; shock, hemorrhage, degree of respiratory failure, degree of postoperative recovery, the manner in which the anesthetic was taken, the duration of the operation, were all factors which could not be accurately controlled; therefore convulsive seizures frequently differed in type, although the procedures were apparently the same.

In two of the three animals with a section below the optic thalami, convulsions were produced that differed in no discernible way from animals with intact central nervous systems. The muscles of the face, jaw, and eyeballs participated in the convulsion. In the remaining animal the convulsions were tonic in character and were followed by running movements. Convulsive movements occurred in the face, jaw and eyeballs as in the other two animals.

It was observed that when a section was made at lower levels in the brain stem, convulsions were produced with greater difficulty; they required at times twice the amount and occasionally even more than twice the amount, of picrotoxin solution. When convulsions were produced, their intensity was far less than those produced in animals in which higher transections had been made. In two of the four animals transected immediately below the level of the nucleus of the seventh cranial nerve, a generalized picrotoxin convulsion was produced. In one of these the muscles of the jaw, face and eyeballs participated in the convulsion. Of the three animals transected at two levels, the animal which received a hypodermic injection of picrotoxin solution following the section immediately above the level of the seventh nerve, developed a typical picrotoxin convulsion with strong clonic contractions of the masseters and of the muscles of the eyeballs. When this animal's brain stem was transected again below the level of the optic thalami, the convulsions became less pronounced, but retained clonic and tonic features, though the movements of the jaw and eyeballs ceased. The remaining two animals did not develop any convulsion after the double transection.

From these animals it will be observed that picrotoxin convulsions can be produced after transection of the brain stem either below or above the optic thalami, or below the level of the seventh cranial nerve, and that these convulsions retain their clonic features. Whether the animal is transected at one or the other level, some muscles of the head and eyeballs participate in the convulsion. In other words, whether the segment innervating the muscles of the face, jaws and eyeballs was connected with the medulla alone or the cerebrum alone, convulsive disorders occurred in these muscles. It seems probable, although not absolutely proved, that this observation indicates the epileptogenous quality of the motor cells in the reticular formation extending from below the optic thalami to the spinal cord. Although it is true that facial movements and ocular movements ceased after complete isolation of such a segment, it could not be shown that the fifth and seventh nerves were uninjured, inasmuch as the conjunctival reflex did not return.

In ten of the frogs I was able to isolate a segment between the optic thalami and a level below the seventh nerve and was able to

produce picrotoxin convulsions with movements of the jaw and of the eyeballs. The movements of the eyeballs in this animal may, however, have been due to respiratory efforts and therefore may not be the result of a convulsion.

It appears that in a frog the jaw participates in a convulsive seizure when the brain stem, from which the fifth nerve originates, has been separated from the rest of the central nervous system. In higher animals, although the influence of the cortex could not be excluded, it seemed probable that epileptogenous qualities existed in the segment that extends from below the optic thalami to the seventh nerve. It was certainly clear that that part of the medulla from the seventh nerve to the spinal cord possessed epileptogenous qualities.

An attempt was made to find a central autonomic nervous system depressant. The only substance to which such a character has been attributed was botulinus toxin. Although animals receiving this toxin, after a period of incubation, which by the administration of large doses was reduced to eight hours, showed very marked respiratory difficulty followed by ataxia and paresis of extremities, the injection of picrotoxin almost always produced picrotoxin convulsions and respiration was in no way affected. It is doubtful whether botulinus toxin is a true central autonomic nervous system depressant.

CONCLUSIONS

From these experiments no conclusive evidence could be adduced that an isolated segment of the brain stem contained epileptogenous elements. It appeared highly probable, however, that such was the case.

It would seem that epileptogenous qualities are possessed by the ganglion cells both of the brain and the brain stem; that convulsive movements may be evoked by proper stimulation of such cells at any level; and that the first symptom produced depends upon the level so stimulated.

STUDIES ON EPILEPSY

I. CONVULSIONS EXPERIMENTALLY PRODUCED IN ANIMALS COMPARED WITH CONVULSIVE STATES IN MAN

A PRELIMINARY REPORT * -

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NEW YORK

Several years ago, a group of physicians under the leadership of Dr. Frederick Tilney, made a critical study of the literature of epilepsy. Frequent meetings were held, and the published papers on the subject were reviewed and discussed. The more the literature was studied, the clearer it became that the nature and cause of the symptoms we call epilepsy, were lost in a maze of theories and contradictions, and that, as a basis for advance, careful research into the causes, nature and site of convulsions was necessary. Only when this is done shall we have a real basis for biochemical research and for logical clinical investigations into the etiology of epilepsy.

With this conclusion came the conviction that the investigation must begin with the simplest problems of the convulsive attack, and gradually advance to the more complex. The following report represents the work along these lines thus far done, but we have touched only the surface of the investigation.

Convulsions in animals can be produced in a variety of ways. After a critical review of previous methods we selected two: first, the injection of essence of absinth (*cultivée*) which was first used by Ossipow and later by many investigators. The second method has, to our knowledge, never before been used for the study of convulsions in animals.¹ We have used methods for the production of convulsions which are perhaps genetically similar to what occurs in man in the convulsive attacks that have a primary toxic or vascular cause. This

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1. We attempted also to use other toxic substances, such as oil of cloves, for injection into the circulation. Our results were inconstant. We have tried to cause uremic convulsions in cats by total or subtotal double nephrectomy, but the results thus far have been negative. Finally we have begun to study the convulsions of asphyxia.

was done because of a certain inherent probability that the ultimate mechanism, and the final pathway of many and perhaps all convulsive attacks in man, no matter what the primary cause or what the disease with which they were associated, were the same.

EXPERIMENTAL METHODS

The experiments were made on adult cats, and, unless otherwise mentioned, the animals received no food for twenty-four hours before they were used.

A. Injections of essence of absinth into the femoral vein.—When essence of absinth (*cultivée*) is injected into the circulation, one or more severe convulsive attacks follow. The convulsions begin abruptly a few seconds after the drug has been injected. There is usually a tonic phase, of short duration, with opisthotonus and retraction of the head, quickly followed by severe clonic convulsions which involve all of the muscles of the body. The convulsions last from one to five minutes, during which the pupils are widely dilated, the corneas are insensitive, respiration is stertorous, and the mucous membranes are cyanotic. Urine is usually expelled and sometimes feces are evacuated. For the ensuing five to ten minutes, there are frequent coarse twitches of the muscles of the trunk and extremities, and sometimes a short period of rigidity of the fore and hind limbs that reminds one of the decerebrate rigidity of Sherrington. After the convulsions have ceased, the animal lies quietly for a time and then sits up and later walks around. Many of the animals, when they first stand up, remain in a fixed position, as if they were afraid to move a single muscle. There may be more than one of the convulsive attacks.

The strength of the solution and the amount to be injected was arrived at only after considerable experimentation. At first we used pure essence of absinth for the injections; later, an emulsion of one part of essence of absinth in twenty of Ringer-Locke solution, of which 0.1 cubic centimeter per pound of animal was injected. We finally found that a solution of one part of essence of absinth in nineteen parts of 95 per cent. alcohol was a useful standard solution with which more exact dosage was possible.² Thirty-five thousandths to 0.05 c.c. of this 5 per cent. solution of absinth in alcohol, per pound of animal, was injected into a femoral vein, and this amount was found to be what one might call a normal convulsing dose. If less, per pound of animal, was injected, convulsions were rarely produced; at most the animal had a few convulsive twitches. If, on the other

2. In control animals 95 per cent. alcohol was injected intravenously but such an injection of alcohol was never followed by a convulsion.

hand, more than 0.06 cubic centimeter per pound of animal was used for the first injection, the cat often died in the first convulsive attack.

In order to make certain that absinth convulsions were cerebral in origin the cerebral circulation was interrupted in the manner that will be described in method B, and during the period of cerebral anemia, absinth was injected into a femoral vein. No convulsions ever occurred. The proof that absinth convulsions were of cerebral origin was thus definitely established.

B. Temporary clamping of the innominate and left subclavian arteries.—This method was suggested by the experiments of Dr. Frank Pike, of the Neurological Department of the College of Physicians and Surgeons, because, in his studies on the respiration, he had observed and reported that convulsions occurred in animals in which he had temporarily shut off the cerebral circulation. We have studied the convulsions caused by cerebral anemia and have developed the method in the manner we shall later describe.

Under intratracheal ether anesthesia, the innominate and left subclavian arteries are exposed and temporary clamps are applied to the vessels, central to the origin of the vertebral arteries. This shuts off all of the blood supply to the brain except an insignificant quantity that reaches the brain through the spinal arteries. In most of the animals the immediate effect of the procedure is the following: A moment after the vessels have been clamped, the animal's body straightens out and respiratory movements cease. The ether anesthesia is at once stopped and artificial respiration begun. This can be easily done with the intratracheal apparatus. During this period of cerebral anemia, the nose and mucous membranes are pallid, the pupils widely dilated, the corneas insensitive, and spontaneous respiratory movements are abolished. Artificial respiration by insufflation of air has to be continued.

When the clamps are removed from the vessels, the nose and mucous membranes become pink again but the pupils remain widely dilated, the corneas insensitive, and respiration is abolished for from five to thirty minutes or more. Finally, the animal begins to make occasional spasmodic inspirations, and, gradually, regular deep respiratory movements are reestablished. The pupils gradually become contracted, and finally the corneal reflexes return. Only then is artificial respiration discontinued and the intratracheal tube removed. We have found that interruption of the cerebral circulation for eleven minutes gave us the best results.

After a period of time, which varied from ten minutes to one hour, tonic and clonic convulsions occur. Stiffness of several or of all of the extremities is first noticed with an increase in the tendon reflexes

of the fore and hind limbs. This is soon followed by tonic and clonic convulsions. The attacks recur a number of times during the period of observation.

- We desire here to call attention to the fact that the convulsions are produced, not by the introduction of a drug into the circulation which may affect every part of the body, but by the simple method of shutting off the blood from the brain. The method permitted us also, in a very simple manner, to establish an artificial circulation through the brain, so that we could pass a variety of fluids through the intracranial circulation. All that was necessary was to inject fluid into a carotid artery above the clamps and to drain off the fluid from one of the jugular veins, a ligature having been tied around the animal's neck and all structures excepting the trachea, esophagus, carotid vessels, and vagus nerves being compressed. This ligature prevents the return of the fluid into the general circulation, except for an amount, that cannot be controlled, that is carried off through the veins in the spinal canal. We thus had a cranial organism—a living test tube for the study of the influence of various solutions and various drugs on the brain. We shall recur in another part of this paper to some of the experiments made with an artificial cerebral circulation, especially to those which have a direct bearing on the subject of this paper. At another time, we shall report upon a number of facts of great interest which have to do with the influence of drugs on the cerebral circulation and upon the functions of the brain.

With these two methods, we have made studies on over 300 cats; as many of these died before experiments were concluded, the number on which this paper is based, is 261 cats. The number of actual experiments was, of course, much larger, as many of the animals were used over and over again.

For the sake of brevity, we shall use the term "convulsing agent" whenever we refer to one of the methods we used for the production of convulsive attacks, and the term "convulsing dose" for the minimum procedure which caused a convulsion in our animals, no matter whether we refer to the duration of the cerebral anemia or the amount of the intravenous injection.

EXPERIMENTAL RESULTS

I. *After one convulsion has been produced, the susceptibility of the brain is much reduced. For the production of a second convulsion the amount of the convulsive agent has to be much increased. In many instances it is impossible to cause a second convulsion no matter how large the "convulsing dose." This increased resistance or diminished susceptibility is probably an exhaustion phenomenon and persists for one to three days, but disappears in less than ten days.*

This series of experiments was made on 42 animals. A convulsing dose of essence of absinth was first injected. Then after intervals which varied from twenty minutes to ten days, the injections were repeated a number of times. In these animals, the intravenous injection of a convulsing dose of essence of absinth was followed, after the expiration of a few seconds, by one or more general convulsions. Occasionally the convulsive attacks began after a somewhat longer period during which the animal showed signs of restlessness. A second injection, given after about twenty to thirty minutes, was not followed by a convulsion, unless the amount of absinth injected was much increased. Even a large dose may not cause a convulsion. The same results were obtained with each succeeding injection. This "refractory" period lasted for several—often for three days, but one week later, the animal's susceptibility was no different from that of an animal that had never received an injection. Some cats are less susceptible to absinth so that the convulsing dose must be larger, and in these animals a convulsion usually followed as soon as a second injection had been given. These animals, also, were thereafter much less susceptible to succeeding injections and their behavior was identical with that of the animals which had a convulsive seizure after the first injection. The explanation for this change in susceptibility is not clear and further investigations along these lines will be made, but, theoretically the phenomenon may be one of exhaustion of some part of the central or peripheral nervous system.

The lack of specific response—a convulsion—to successive convulsing doses is seen also after the temporary interruption of the blood supply to the brain by the second method we have described above (method B) although its demonstration is difficult. If, after a convulsion has been produced by an intravenous injection of absinth, the brain is deprived of its blood supply for eleven minutes by temporary compression of the innominate and left subclavian arteries, no convulsions follow this latter procedure (see Experiments 123 and 134). In other words, if the animal has had a convulsion from absinth, it is less susceptible, or not at all susceptible, to convulsive attacks from cerebral anemia.

SAMPLE PROTOCOLS FROM THIS SERIES OF EXPERIMENTS

PROCEDURE	RESULT
Cat 126. Weight, 6 pounds.	
1. 0.75 c.c. of 5 per cent. emulsion of absinth in Ringer-Locke solution injected into the femoral vein.	Immediate violent convulsion for five minutes.
2. One hour later: Injection of 1.2 c.c.	No convulsion.
Cat 127. Weight, 5 pounds.	
1. 0.5 c.c. of absinth in Ringer-Locke solution injected (0.1 c.c. per pound).	Immediate violent convulsion for five minutes.

PROCEDURE	RESULT
2. Ninety minutes later: Second injection. 1 c.c. (0.2 c.c. per pound).	Delayed convulsion of short duration.
Cat 128. Weight, 5 pounds.	
1. 0.5 c.c. (0.1 c.c. per pound) of absinth in Ringer-Locke solution injected.	Immediate violent convulsion for two minutes.
2. Sixty minutes later: Injection of 0.5 c.c.	No convulsion.
3. Thirty minutes later: Injection of 1 c.c. (0.2 c.c. per pound).	Delayed convulsion, not violent nor of long duration.
Cat 144. Weight, 7¾ pounds.	
1. Injection of 0.75 c.c. of 5 per cent. solution of absinth in 95 per cent. alcohol.	Violent convulsion for one minute. After three minutes violent convulsion for two minutes.
2. Forty-five minutes later: Injection of 0.4 c.c.	No convulsion.
3. One week later: Injection of 1.5 c.c.	Two violent convulsions.
4. Fifteen minutes later: Injection of 1.5 c.c.	No convulsion.
Cat 146. Weight, 8 pounds.	
1. Injection of 0.4 c.c. (0.05 c.c. per pound) of absinth in alcohol.	Immediate violent convulsion.
2. Thirty-five minutes later: Injection of 0.8 c.c. (0.1 c.c. per pound).	Delayed violent convulsion.
Cat 148. Weight, 7 pounds.	
1. Injection of 0.35 c.c. (0.05 c.c. per pound) of absinth in alcohol.	Two violent convulsions in close succession followed by many muscular twitches.
2. Fifteen minutes later: Injection of 0.35 c.c.	No convulsion or twitchings.
Cat 230. Weight, 8 pounds.	
4:05 p. m.: Injection of 0.24 c.c. of absinth in alcohol (0.03 c.c. per pound).	No convulsion.
4:31 p. m.: Injection of 0.32 c.c. (0.04 c.c. per pound).	Convulsion for two minutes.
4:50 p. m.: Injection of 0.32 c.c.	No convulsion.
Cat 233. Weight, 7¼ pounds.	
3:10 p. m.: Injection of 0.29 c.c. (0.04 c.c. per pound) of absinth in alcohol.	Immediate violent convulsion and prolonged muscular twitches.
3:53 p. m.: Injection of 0.29 c.c.	No convulsion.
4:20 p. m.: Injection of 0.35 c.c. (0.05 c.c. per pound).	No convulsion.
4:43 p. m.: Injection of 0.43 c.c. (0.06 c.c. per pound).	No convulsion.
Cat 234. Weight, 5¼ pounds.	
3:17 p. m.: Injection of 0.21 c.c. of absinth in alcohol (0.04 c.c. per pound).	Immediate violent convulsion for one minute followed by muscular twitches for fifteen minutes.
4:25 p. m.: Injection of 0.26 c.c. (0.05 c.c. per pound).	No convulsion.
4:46 p. m.: Injection of 0.31 c.c. (0.06 c.c. per pound).	No convulsion.
Cat 248. Weight, 5½ pounds.	
3:55 p. m.: Injection of 0.25 c.c. of absinth in alcohol (0.05 c.c. per pound).	Two severe convulsions.
4:10 p. m.: Injection of 0.25 c.c.	No convulsion.

PROCEDURE	RESULT
5:15 p. m.: Injection of 0.25 c.c.	Few muscular twitches.
Cat 123. Weight, 10¾ pounds.	
1. 0.1 c.c. of essence of absinth.	Violent convulsion for five minutes.
2. Ninety minutes later, clamping of cerebral vessels for eleven minutes.	No convulsion.
Cat 134. Weight, 5 pounds.	
1. Injection of 0.5 c.c. of absinth in Ringer-Locke solution.	No convulsion.
2. Fifteen minutes later: Injection of 0.5 c.c.	Violent convulsion.
3. Two and one-half hours later: Clamping of cerebral vessels for ten and one-half minutes.	No convulsion.

II. *In thyroidectomized animals, the susceptibility to the first injection of absinth is greater than in normal animals, and the susceptibility to repeated injections is completely altered. Each injection of absinth causes a convulsion.*

This series of experiments was performed upon twelve cats in which the thyroid had been completely removed, and which had survived for seven to fourteen days.³ The experiments demonstrated the remarkable fact that the animals were more susceptible than normal cats to convulsive attacks after the first convulsing dose of absinth, and that they were susceptible to repeated injections, each injection being followed by a violent convulsion and prolonged muscular twitchings. Some typical experiments follow:

PROCEDURE	RESULT
Cat 222. Weight, 5¾ pounds. Thyroidectomy seven days previously.	
2:50 p. m.: Injection of 0.06 c.c. of absinth in alcohol (0.01 c.c. per pound).	Short convulsion after one minute.
3:02 p. m.: Injection of 0.12 c.c. (0.02 c.c. per pound).	Violent convulsion for one and one-half minutes.
Cat 223. Weight, 5½ pounds. Thyroidectomy seven days previously.	
2:35 p. m.: Injection of 0.17 c.c. of absinth in alcohol (0.03 c.c. per pound).	Violent convulsion after five seconds.
Cat 245. Weight, 5½ pounds. Thyroidectomy twelve days previously.	
2:46 p. m.: Injection of 0.16 c.c. of absinth in alcohol (0.03 c.c. per pound).	Rapid breathing.
3:07 p. m.: Injection of 0.3 c.c. of absinth in alcohol (0.05 c.c. per pound).	Immediate violent convulsion followed by muscular twitches.
3:30 p. m.: Injection of 0.30 c.c. (0.05 c.c. per pound).	Violent convulsion with twitches.

3. As the parathyroid glands in cats, and the blood vessels which supply them, are very small, we have not yet been able to determine whether all of the parathyroids were injured during the thyroidectomies.

PROCEDURE	RESULT
3:50 p. m.: Injection of 0.3 c.c.	Violent convulsion for two minutes.
4:11 p. m.: Injection of 0.3 c.c.	Three severe convulsions followed by muscular twitches.
4:31 p. m.: Injection of 0.3 c.c.	Died.
Cat 246. Weight, 10 pounds. Thyroidectomy one week previously.	
2:57 p. m.: Injection of 0.5 c.c. of absinth in alcohol (0.05 c.c. per pound).	Immediate violent convulsion.
3:25 p. m.: Injection of 0.5 c.c.	Violent convulsion.
3:47 p. m.: Injection of 0.5 c.c.	Violent convulsion.
Cat 247. Weight, 5 pounds. Thyroidectomy one week previously.	
2:33 p. m.: Injection of 0.15 c.c. of absinth in alcohol (0.03 c.c. per pound).	Two convulsions after two minutes, followed by muscular twitches.
3:12 p. m.: Injection of 0.15 c.c.	Severe twitching of muscles of body and extremities.
3:36 p. m.: Injection of 0.15 c.c.	Violent convulsion.
4:04 p. m.: Injection of 0.15 c.c.	Violent convulsion.
Cat 261. Weight, 3½ pounds. Thyroidectomy one week previously.	
12:22 p. m.: Injection of 0.17 c.c. of absinth into femoral vein (0.05 c.c. per pound).	Six violent convulsions with twitches during twenty minutes.
1:47 p. m.: Injection of 0.15 c.c. absinth.	Six violent convulsions during eighteen minutes.
3:02 p. m.: Injection of 0.15 c.c. absinth	Seven violent convulsions during thirty-eight minutes.

Of the twelve cats used for this series, eight gave positive results; two died after the second injection, and two gave negative results. More than one convulsion after each absinth injection was often seen; one of the animals (Cat No. 261) had six or seven severe convulsive attacks after each absinth injection.

The results of these experiments suggest that the thyroid (and perhaps the parathyroid glands) has a profound influence on the parts of the brain, and possibly of the peripheral nervous system, concerned in the convulsive attacks, and perhaps has significance with respect to the frequency of convulsive attacks in man. It may be that the thyroid (and perhaps other ductless glands) exerts an inhibitory or check influence upon the activity of the cortical and subcortical motor cells and even on other parts of the motor pathways; the experiments just described seem to point in that direction. Lack of thyroid gland activity may play an important rôle in so-called status epilepticus.

We have made some investigations upon the effect of absinth in animals who had received an intravenous injection of thyroid extract. These experiments are as yet few in number, but they seem to indicate that cats who have been given an intravenous injection of thyroid extract, followed by a convulsing dose of absinth, react in the same way as normal animals. We have not yet investigated the susceptibility

of thyroidectomized cats to convulsions from cerebral anemia produced by our method B, nor have we given injections of thyroid extract followed by absinth to cats from whom the thyroid gland had previously been removed. These experiments will be done in the near future.

III. *Diminished alkalinity of the blood does not render cats more susceptible to convulsions produced either by injections of absinth or by cerebral anemia. It is possible, therefore, that the diminished alkalinity of the blood so often found in man before and during an epileptic attack occurs as a result of, or in association with, the convulsive attack, rather than that it favors a convulsive seizure.*

This conclusion was arrived at as the result of experiments on forty-six cats in which the alkalinity of the blood was diminished by means of injections of lactic acid; in many of them this was done before and after convulsing agents were used. The alkaline reserve in the tissues of the body, is continuously yielded to the blood so that the reaction of the blood remains almost constant. When the alkaline reserve has been used up, the blood becomes acid and death at once ensues. A large number of experiments was required before we had determined the amount of acid necessary to overcome the alkaline reserve and reduce the alkalinity of the blood without causing the death of the animal. We finally determined that an injection of 1 c.c. of a 10 per cent. solution of lactic acid, U. S. P., for every pound of animal would reduce the alkalinity of the blood for a short period. The tests were made with the Van Slyke carbon dioxid method, and the Van Slyke apparatus, and the following results were obtained:

Cat 151. Weight, 9 pounds. Under intratracheal anesthesia, 7 c.c. of blood was extracted from a vein and the gas content of the plasma was determined by the Van Slyke method. The reading of the scale was 5. Five minutes later, 9 c.c. of 10 per cent. lactic acid was injected into the jugular vein. Twenty minutes later, blood was extracted and tested. The reading was 4.2.

Cat 152. Weight, $6\frac{1}{4}$ pounds. The gas content of the blood plasma before the injection at three tests was 5, 5, and 5. The gas content of the blood plasma twenty minutes after injection of 6.25 c.c. of 10 per cent. lactic acid was 4, 4 and 4.2 in three tests.

These experiments demonstrated that the blood alkalinity had been reduced by the amount of acid injected.

We then attempted to learn whether animals, in which the alkalinity of the blood had been reduced, were more susceptible to injections of absinth, so that the convulsing dose could be smaller than in normal cats. As the experiments demonstrate, diminished alkalinity of the blood has no such effect.

PROCEDURE	RESULT
Cat 156. Weight, 5¾ pounds.	
1. Intravenous injection of 5.75 c.c. of 10 per cent. lactic acid (1 c.c. per pound).	
2. One minute later: Injection of 0.28 c.c. (0.05 c.c. per pound) of a solution of absinth 1 part and 95 per cent. alcohol 19 parts.	No convulsion.
3. Forty-five minutes later: Injection of 0.28 c.c. of absinth solution.	Violent convulsion for one minute.
Cat 157. Weight, 5½ pounds.	
1. Injection of 5.5 c.c. of lactic acid solution.	
2. One minute later: Injection of 0.27 c.c. of absinth.	No convulsion.
3. Forty-five minutes later: Injection of 0.22 c.c. of absinth (0.04 c.c. per pound).	No convulsion.
Cat 159. Weight, 6¼ pounds.	
1. Injection of 6.25 c.c. of lactic acid solution (1 c.c. per pound).	
2. One minute later: Injection of 0.31 c.c. (0.05 c.c. per pound) of absinth.	Delayed convulsion which lasted one minute.
3. Forty-five minutes later: Injection of 0.31 c.c. of absinth.	No convulsion.
Cat 161. Weight, 7½ pounds.	
1. Injection of 7.5 c.c. of lactic acid solution.	
2. One minute later: Injection of 0.3 c.c. (0.04 c.c. per pound) of absinth.	No convulsion.
3. Thirty minutes later: Injection of 0.45 c.c. (0.05 c.c. per pound) of absinth.	Two violent convulsions.
Cat 186. Weight, 8½ pounds.	
1. Injection of 17 c.c. (2 c.c. per pound) of lactic acid solution.	
2. A few seconds later: Injection of 0.21 c.c. (0.025 c.c. per pound) of absinth.	No convulsion.

These few experiments, typical of the protocols of many, demonstrate that there is no increased susceptibility to convulsions by absinth when the alkalinity of the blood has been decreased. In fact six of fifteen cats which had received an injection of lactic acid followed by a normal convulsing dose of the solution of absinth, (0.035 to 0.05 c.c. per pound of animal) did not have any convulsion at all, and five others had only muscular twitchings. If any inference could be drawn from these eleven out of fifteen experiments, it would be that cats in which the blood alkalinity has been reduced by lactic acid, are less susceptible to absinth convulsions than are normal cats. It is well to remember, in this connection, that alkalinity of the blood artificially produced, is a condition entirely different from one in which the reduction has occurred as the result of disturbances or changes in metabolism.

In an additional series of experiments, the animals were given less than a normal "convulsing dose" of absinth, followed by an injection

of the diluted lactic acid. The object of these experiments was to determine whether a convulsion would follow diminution of the blood alkalinity. As shown in the accompanying table, not one of these animals had a convulsion after the diluted lactic acid had been injected and the blood alkalinity thereby reduced. The experiments of this

ABSINTH INJECTIONS AFTER REDUCTION OF BLOOD ALKALINITY

Cat No.	Weight in Pounds	Amount of Absinth Injected, C.c.	Cubic centi- meters per Pound	Result	Cubic centimeters Lactic Acid 1 to 3 Minutes Later	Result
162.....	6.75	0.27	0.04	Convulsion	6.75	0
163.....	5.25	0.18	0.035	Convulsion	5.25	0
164.....	6.50	0.16	0.035	Convulsion	6.50	0
165.....	5.50	0.08	0.015	0	5.50	0
166.....	5.50	0.08	0.015	0	5.50	0
167.....	9.00	0.18	0.02	0	9.00	0
170.....	4.50	0.11	0.025	0	4.50	0

series show that diminished alkalinity does not favor, nor make an animal more susceptible to, a convulsive attack from absinth but, on the contrary, that diminished alkalinity as such makes the animal rather more resistant. These results suggest that the change in the alkaline reaction of the blood before convulsive attacks in man, found by many investigators, is merely associated with some other factor, and has no etiologic relation to, and does not increase, the susceptibility of the individual to a convulsive attack.

IV. *Animals that have been starved for seventy-two hours are not less susceptible to absinth convulsions than those that have been fed shortly before the injections.*

As has already been mentioned, most of the cats we used did not receive any food for twenty-four hours before the experiments. Of the fourteen cats of series IV, six were fed a few hours before they were used, and eight had been without food for seventy-two hours. The following are a few of the experiments of each group:

PROCEDURE	RESULT
Cat 228. Weight, 6½ pounds. Fed a few hours previously.	
3:54 p. m.: Injection of 0.2 c.c. of absinth in alcohol (0.03 c.c. per pound).	Twitching and rapid breathing.
4:18 p. m.: Injection of 0.26 c.c. (0.04 c.c. per pound).	Convulsion lasting two minutes; twitching.
4:40 p. m.: Injection of 0.26 c.c.	Occasional twitch.
Cat 232. Weight, 5½ pounds. Fed a few hours previously.	
3:04 p. m.: Injection of 0.16 c.c. of absinth in alcohol (0.03 c.c. per pound).	No convulsion.
3:47 p. m.: Injection of 0.16 c.c.	One twitch.

PROCEDURE	RESULT
4:17 p. m.: Injection of 0.27 c.c. (0.05 c.c. per pound).	Violent convulsion.
4:40 p. m.: Injection of 0.33 c.c. (0.06 c.c. per pound).	Occasional twitch.
Cat 236. Weight, 5 pounds. Fed a few hours previously.	
3:41 p. m.: Injection of 0.17 c.c. of absinth in alcohol (0.035 c.c. per pound).	Violent convulsion.
4:07 p. m.: Injection of 0.2 c.c. (0.04 c.c. per pound).	No convulsion.
4:34 p. m.: Injection of 0.25 c.c. (0.05 c.c. per pound).	No convulsion.
Cat 231. Weight, 7½ pounds. No food for seventy-two hours.	
2:59 p. m.: Injection of 0.22 c.c. of absinth in alcohol (0.03 c.c. per pound).	One slight twitch.
3:40 p. m.: Injection of 0.22 c.c.	One twitch.
4:12 p. m.: Injection of 0.37 c.c. (0.05 c.c. per pound).	Violent convulsion with twitches.
Cat 235. Weight, 5¾ pounds. No food for seventy-two hours.	
3:27 p. m.: Injection of 0.20 c.c. of absinth in alcohol (0.035 c.c. per pound).	Immediate convulsion.
4:04 p. m.: Injection of 0.23 c.c. (0.04 c.c. per pound).	No convulsion.
4:31 p. m.: Injection of 0.29 c.c. (0.05 c.c. per pound).	No convulsion.
Cat 237. Weight, 6¼ pounds. No food for seventy-two hours.	
5:04 p. m.: Injection of 0.19 c.c. of absinth in alcohol (0.03 c.c. per pound).	One twitch.
5:15 p. m.: Injection of 0.31 c.c. (0.05 c.c. per pound).	Very severe convulsion for one minute.

Especially during the past few years, the claim frequently has been made, and there seems to be considerable clinical evidence to support it, that in so-called epileptics, convulsive attacks occur less frequently if the patients are kept on a low protein diet or are actually starved. In this connection, the results of the experiments of this series give food for thought, for they show that, in animals, the susceptibility to absinth convulsions is not influenced by starvation.

V. The prevention of the convulsive attacks which follow temporary cerebral anemia by an artificial cerebral circulation during the period of compression of the innominate and subclavian arteries.

For this series of experiments 107 cats were used. In twenty-six of the animals the nature of the convulsive attacks which follow the cerebral anemia, produced by temporary compression of the innominate and left subclavian arteries, was investigated. In eighty-one cats, an artificial cerebral circulation was established in the following manner: Just before the blood vessels were clamped, a needle was inserted into the left carotid artery and fluid was allowed to run in slowly. The

clamps were then applied to the vessels, and a tape which did not include the trachea, esophagus, carotid arteries or vagus nerves, was tightly tied around the animal's neck. This ligature prevented any of the fluid which was passing through the cerebral blood vessels (excepting a small amount which passed through the veins in the spinal canal and which could not be controlled) from entering the general circulation.

A small opening was quickly made in the right jugular vein so that the injected fluid could escape, or a glass canula was inserted into the vein so that the fluid could be collected and the amount measured. In this way, an artificial cerebral circulation was established. The fluid entered through one carotid artery, circulated through the blood vessels of the brain and escaped through the jugular vein of the opposite side. This artificial circulation was continued during the entire period of the cerebral anemia. The needle in the artery was removed and the canula withdrawn from the jugular vein, just before the clamps were removed from the large arteries of the neck.

a. Animals in whom normal saline solution or Ringer-Locke solution is allowed to circulate through the vessels of the brain during the period of cerebral anemia, do not have any convulsions after the intracranial circulation is reestablished.

In order to understand the significance of the above mentioned facts, the reader must remember that convulsions regularly follow the cerebral anemia produced by the temporary clamping of the large vessels of the neck, as described in method B. When, however, this artificial circulation of saline or Ringer-Locke solution is established for the period of the cerebral anemia, convulsions rarely occur. In a few instances, in which the large arteries in the neck were probably unduly traumatized by the clamps, so that clotting occurred, there were slight convulsive seizures, but these cats formed the exception. In the large majority of the experiments, convulsions did not occur, and the animals behaved like normal cats as soon as they had recovered from the ether.

During the period of the cerebral anemia and of the artificial cerebral circulation, most of the animals had to be kept alive by artificial respiration; in some of them respiratory movements returned three to five minutes after the artificial cerebral circulation of saline or Ringer-Locke solution had been established.

This freedom from the convulsions of cerebral anemia, must have been due to one of three conditions: Either the solution injured the motor cells or conducting fibers in the brain, so that no excitatory impulses could be sent out to the muscles, or the saline or Ringer-Locke solution was an efficient substitute for blood for the period

during which it was used; or, finally, the solution used, removed some product of disturbed metabolism which was excitatory for the motor cells. We have not yet been able to determine which of the two last mentioned possibilities is the correct one. It is certain, however, that the saline and Ringer-Locke solutions did not damage and therefore interfere with the excitability of the motor cells. This was proved by the fact that the animals remained susceptible to absinth, so that the injection of a "convulsing dose" of absinth was promptly followed by a typical convulsion.

b. *Hypotonic and hypertonic saline and Ringer-Locke solutions also prevent the convulsive attacks which follow temporary cerebral anemia.*

In these experiments, the salt content of the solutions used for the artificial intracranial circulation was diminished or increased in successive series of animals. Finally, pure distilled water was used at one end and 2 per cent. of salt at the other end of the scale. These variations in the concentration of the solutions used, made no essential difference in the results of the experiments. If the animals survived, they did not have any convulsive attacks. When distilled water was used, hemolysis often occurred and was followed in one-half to one hour by the death of the animal. Two per cent. saline or Ringer-Locke with 2 per cent. of salt usually had a profound effect upon the respiratory center, so that respiratory movements were never reestablished.

CONCLUSIONS

The experiments described in this paper represent the beginning of our attempt to learn more of the nature of convulsive attacks. Results of experiments must always be applied with some reserve to conditions in the human being, for in man symptoms are a result of a disease process, while in animals symptoms are produced by artificial means. The results of our experiments are, with this reservation, suggestive.

METABOLIC STATES CONTRIBUTING TO UREMIA *

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The term uremia has been employed in the last few years with two different meanings: to designate a syndrome of symptoms and signs referable to the central nervous system and associated with nephritis; also to designate an increase of the urea in the blood above the normal concentration. In this paper the term will be used in its older sense, namely to designate a state characterized by psychic and motor disorders developing in some cases of nephritis.

Chronic nephritis is a disease of long duration involving not only the kidneys but also the heart, arteries, and possibly other organs. With the number of factors possibly contributing to coma, for example, the problem becomes too complex for direct investigation. On this account uremia developing in cases of acute nephritis offers a better opportunity for study. First the patients are often young and on this account extensive degenerative changes, especially of the heart and arteries are less likely to have occurred. Next the period of duration of the disease is brief, often a matter of days only. And if lesions occur in other organs than the kidneys they are not usually advanced in degree or pronounced in character. The study of cases of acute nephritis and uremia then promises most toward solving the problem of the causation of uremia. Acute nephritis is usually a sequel of some type of infection but often the primary infection has subsided before the first sign of renal disorder appears.

The fact that arrests attention in all types of nephritis is some sort of impairment of excretion. It is this decrease of excretion which measures the degree of severity of the renal disorder, and it matters little in the first instance whether it be water, salt, or some nitrogen compound that fails of excretion. These differences demark types of functional disorder primarily and also, of course, the extent of severity of the lesion.

As a consequence of disordered excretion there results an accumulation of waste products in the organism—retention; when there is retention of water and sodium chlorid, this leads to edema; when of phosphorus, to acidosis in some instances. It was natural to search among waste substances retained in blood and tissues for some one substance which might be held directly responsible for the most striking type of

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uremia, the convulsive type. Some of the known nitrogen compounds excreted in the urine have at some time or other been examined for specific toxicity and tested to determine whether or no, in adequate dosage, any one of them might cause convulsions in animals. The results of experiment have not been decisive. And there is moreover, an insurmountable obstacle to the idea that simple retention of normal waste products can induce uremia. This obstacle is the fact that absolute retention caused either by mercury poisoning, or by the removal of the kidneys in man is not followed by uremia. Complete anuria resulting from bichlorid of mercury poisoning is not a rare occurrence and we have had opportunity to observe two cases of anuria caused by the removal of the only functioning kidney. There are several such cases reported in the literature. The striking fact is that in cases of this sort there is an enormous accumulation of nitrogen waste products in the body. Determinations of urea and nonprotein nitrogen in the blood give figures higher than those found in the last stage of nephritis, yet these patients develop none of the evidences of uremia. From the experimental side too, comes only negative evidence. Removal of the kidneys in animals, or ligation of the renal vessels or of the ureters produces no characteristic symptom other than the retention shown in chemical analyses of the blood. There is then, no alternative to the conclusion that simple retention is not the immediate cause of uremia, although retention (defective elimination) is the most clear cut consequence of renal disease. There must occur in nephritis a production of some substance foreign to normal catabolism; or the disease nephritis must so change the nervous tissues that they become increasingly sensitive to normal metabolites.

The retention of some chemical compounds at least is not peculiar to nephritis. Thus for example, the retention of sodium chlorid is a conspicuous abnormality in some types of nephritis, but salt retention is notable also in rheumatic fever and pneumonia. There is this difference, however, in the modes of retention in the diseases mentioned. When sodium chlorid is retained in nephritis there is commonly water retention as well and this water retention leads to edema and anasarca. A different state of affairs prevails in the salt retention of febrile diseases. In pneumonia, for example, the sodium chlorid excretion in the urine is only minimal during the febrile period; yet there is no significant water retention during this time. In nephritis, on the other hand, when sodium chlorid is retained there is simultaneous water retention. And the relation of the quantity of salt retained to the amount of water retained seems to be definite and in mathematical. This relation is that of the physiologic solution. The experiment of measuring rapid gains in body weight after the ingestion by nephritics with edema of definite amounts of salt has often been made. This gain in

weight is due to retained water and can be predicted often by computation from the salt retention. The retained salt may not be detectable as an excess of chlorids in the plasma, on account of this water retention. Sometimes, however, there is excess of chlorid in blood plasma or blood corpuscles. And the determining factor as to whether the excess of chlorid is in the plasma or the cells seems to be the hydrogen-ion concentration of the blood. Probably this is true for tissue cells as well as blood cells. The probabilities are that the retained chlorid is not in solution, as we ordinarily understand the term, but rather in union with body colloids; as a result, the colloids imbibe more water than normally. We must leave this question here in an unsettled state since our knowledge of colloids is too imperfect to enable us to understand clearly the production of edema. It is not demonstrated as yet for all types of renal disease whether failure of excretion of sodium chlorid is due primarily to inability of the kidney to excrete (as seems the case when plasma chlorid is high) or whether the chlorid is held in a colloidal combination which withholds it from renal filtration. The fact worthy of note is that the retention in renal disease is not a simple retention, but is associated with profound changes in the body chemistry, the cell colloids, and results in water retention and edema.

The subject of edema is fundamental in many problems of neurology. Nephritic edema represents one type and is important in this discussion since edema of the brain is a conspicuous lesion in one kind of uremia. Apropos of the general subject of edema, some basic principles are worthy of note since they have a wide application in disorders of the central nervous system. When the blood supply of a cell is interrupted, a chemical change is at once initiated in that cell. The deprivation of oxygen causes first cellular acidosis and, as a consequence of this reaction, the colloids imbibe water and the cell swells—becomes edematous. This sort of disorder finds examples not only in heart disease, but in circumscribed disease of blood vessels even though the interruption in the circulation is but temporary. One sees then that the same lesion in tissues may be produced in quite different ways. Edema is a colloid change but it may be induced by an excess of salt consequent to renal retention, or it may be induced by cellular asphyxia resulting from a disease in the circulation. This very brief sketch of two contrasting types of edema suggests the reason for similarity in the clinical manifestations between the coma due to nephritic salt retention and the coma of arteriosclerosis.

The second inorganic substance that is sometimes important in uremia is phosphorus. Like chlorid salts the phosphates fail of normal excretion at times and are retained in the tissues. This retention of phosphates leads to acidosis which is sometimes, though rarely, a conspicuous symptom in uremia. The existence of acidosis is demon-

strated by the low hydrogen-ion concentration of the blood and further by therapeutic observation that administration of adequate alkalis relieves alike the symptoms and the signs of acidosis. When it is recalled that the phosphates are the chief agents for maintaining normal reaction in the body fluid it becomes evident how quickly any disorder of phosphate metabolism will declare itself in clinical symptoms.

As explained, none of the nitrogenous waste products found in normal urine can be held directly responsible for convulsive uremia, but the subject of retention of these waste products demands a little further examination. First there is a difference in the facility with which the various known compounds are excreted by the diseased kidney. For example, there may be a definite increase of uric acid in the blood plasma at times when urea and other nitrogenous bodies seem to be normally excreted. This fact has given rise to the idea of selective excretion. A good example of this differentiation of renal function is often noted in chronic nephritis when there is no retention of salt, while at the same time the urea retention is of such a degree that the blood urea is fourfold the normal.

If one determine the amounts of the various nitrogen-containing substances in the urine, e.g., urea, ammonia, uric acid, creatinin, amino-acid, it is found that the sum derived by adding the amounts of nitrogen in these several fractions together just about equals the total nitrogen of the urine. But if we determine the total nonprotein nitrogen in blood, and then the various fractions of which it is composed, the sum of the known fractions does not equal the total. In other words there are nitrogen-containing substances in blood whose character is at present not known, while we know apparently all of the substances in urine. This unknown nitrogen we speak of as the "undetermined" or "rest" nitrogen. Normally the undetermined nitrogen in blood is somewhere about twenty per cent. of the total, but in uremia it is often nearly half. Two possible interpretations suggest themselves in explanation, either the undetermined nitrogen fraction in nephritis finds more difficulty of excretion than any other fraction, or it is produced in excess. Both might conceivably be true. This undetermined nitrogen of blood is doubtless made up of several substances which will eventually be isolated and identified. One of these substances we have thought to be creatinin because of certain reactions, but it is now known that there is no significant amount of creatinin in blood. The behavior of this creatinin-like substance is interesting because in uremic states it increases out of all proportion to other nitrogen waste products. Moreover when there is considerable increase of this *x*-substance, uremia may be predicted.

Since the undetermined nitrogen fraction in urine is very small, in normal blood somewhat larger, and in uremic blood mounts up to thirty or forty per cent., it seems probable that this fraction normally under-

goes transformation into some known waste product; but in severe nephritis and uremia this transformation is slow and imperfect. Where the chemical change occurs and what are the products of this change are not at present known.

Thus far attention has been given to those alterations of the composition of body fluids which result from failure of the kidney as an excretory organ; that is to say to the consequences of retention of waste products. But the kidney is not alone an excretory organ. It is apparently in a sense analytic since it excretes selectively. Whether it is actually synthetic we are not sure. The production of hippuric acid from benzoic acid might be so regarded, but we are not certain that the whole process occurs in the kidney; the liver may be concerned as well.

Ammonia apparently is a product of renal function purely. Formerly we thought of the liver and possibly the muscles as sources of ammonia, but this is no longer the case. If it be recalled that many of the products of normal cell activity are acid in character, and that in various disease states there is an excessive production of acids, then the importance of a source of available alkali in the body can be appreciated. In what degree the failure of ammonia formation contributes to the acidosis of uremic states must be left unanswered. The retention of phosphates already mentioned would conceivably be more rapid in producing clinical effect if combined with a failure of ammonia as a neutralizing agent.

I have endeavored to describe some of the more conspicuous disorders in metabolism which contribute to uremic states. It is the way in which these several disorders are combined that determines the clinical picture and the type of uremia. Retention in nephritis is never pure retention, but is always associated with profound readjustments in the body chemistry. Convulsive uremia, for example, is usually demarked by considerable nitrogen retention, but this is not without exception. In this type of uremia, there seems to be a specific toxic base in the blood; this base has been isolated though never identified. It is conceivable on the basis of analogy that this substance results from a reversal of chemical reaction. In diabetes butyric acid is transformed into hydroxybutyric acid, then into diacetic acid. If for some reason the diacetic acid fails of prompt excretion and accumulates in the body, there results a reversal of the process and the diacetic acid is transformed back into hydroxybutyric acid. So in nephritis a normal process may be reversed by failure of excretion. There are many substances formed in metabolism which are powerful toxins; no effects occur ordinarily because the toxin is promptly transformed into some other substance. But if at the final stage the product were not excreted,

there would at once ensue an increase of all the precursors of these substances, the toxins among them.

All of these various factors with which we have been dealing might be designated as direct factors in the production of uremia, since all of them result from the primary systemic disorder which we call nephritis. Besides these direct factors and most important among them is the state of the circulation. In chronic nephritis, degeneration of the arteries and myocardium is of such frequent occurrence that we often speak of cardiorenal disease, or vascularrenal disease. The vascular hypertension of nephritis is an added strain imposed on a laboring heart so that myocardial insufficiency is a complication of common occurrence. The normal renal function depends primarily on the integrity of the circulation through the kidney. If myocardial weakness induces stasis, renal function is at once impaired. Sometimes cases of nephritis come under observation at a time when the amount of functioning renal tissue seems just sufficient to meet the demands of the individual. The normal latitude to increased demands has gone, conditions are in a state of equilibrium in a restricted environment. Now if there develops myocardial insufficiency with its consequent drop in renal function, uremia develops. In this sense we may quite correctly think of the heart as the immediate agent in precipitating the uremic state.

Nephritis is a constitutional disease, often with pronounced heritable predisposition. The renal lesions and impairments of function are the conspicuous signs; but probably more important are the changes in metabolism which accompany or precede impairment of excretion. Simple retention of excretory substances is the result of lowered renal function; this retention induces profound changes in the body colloids primarily. The delicate adjustment of acid-base equilibrium in the body may be upset by acid retention and by failure of ammonia formation. At times specific toxins are formed, due possibly to a reversal of chemical reactions.

News and Comment

THE STATUS OF EPIDEMIC ENCEPHALITIS AS AN INDEPENDENT DISEASE

From the spring of 1920 until recently, there have been comparatively few new cases of epidemic (lethargic) encephalitis in North America, and interest in it has been kept up mainly by the surprising array of its somatic and psychic sequels. Within the last few weeks, however, reports have come of a large epidemic in Winnipeg, and smaller ones in Connecticut and elsewhere. Since 1917, nearly 2,000 articles on this disease have appeared, and, within the last two years, four comprehensive reviews in book form.¹ In addition, the French investigator Levaditi has written a book giving a comparative epidemiologic, pathologic and clinical study of the three closely related acute infectious disorders of the nervous system: encephalitis, poliomyelitis and herpes.²

The etiology of both acute encephalitis and poliomyelitis remains unsettled. The champions of the "globoid bodies" of Loewe, Hirschfeld and Strauss, and those of the streptococcus, have not yet agreed among themselves, or succeeded in convincing the majority of investigators to accept any exclusive view. It is agreed that the disease is caused by a microbe having a minute filtrable form that permits transmission by means of the filtrable virus to animals. It seems proved also that the virus exists in the nasopharynx, that the disease is mildly contagious, and that the usual period of incubation is ten days. No one now regards seriously the view of the English epidemiologist Crookshank that epidemic encephalitis and poliomyelitis are caused by the same virus or variations of the same virus. The relationship between influenza and encephalitis is most difficult to define. It does not seem reasonable that the appearance of the two diseases in epidemic form at the same time now and in the past can be a mere coincidence. There must be some connection, but what is it? Encephalitis is certainly not a mere sequel of influenza, as most victims of the former do not give a history of having had the latter. Nor do encephalitis patients present the leukopenia and tendency to a peculiar form of pneumonia so characteristic of influenza. Furthermore, there is a form of encephalitis directly connected with influenza, which differs from the epidemic form in being more sudden and severe in its onset, and in having more severe and permanent focal symptoms, more signs of a complicating meningitis, and a marked tendency to multiple minute hemorrhages in the brain and sometimes

1. Acute Epidemic Encephalitis: An Investigation by the Association for Research in Nervous and Mental Disease, New York, 1921. Encephalitis Lethargica, Reports on Public Health and Medical Subjects, No. 11, London 1922. Achard, Charles: L'encéphalite léthargique, Paris, 1921. Stern, Felix: Die epidemische Encephalitis, Berlin, 1922.

2. Levaditi, C.: Ectodermoses neurotropes: poliomyélite, encéphalite, herpès, Paris, 1922.

to suppuration. Economo has suggested that the encephalitis virus requires activation by that of influenza, and it has also been thought that the damage to vessel walls caused by previous influenza renders a person more susceptible to encephalitis. Another theory, which at least cannot be said to have been positively disproved, is to the effect that encephalitis is caused by a highly neurotropic influenza virus. Stern offers the broad hypothesis that encephalitis is caused by a filtrable virus, which exists in a harmless form in the saliva of many persons, and may be activated not only by the influenza virus, but also by pneumococci, streptococci and other bacteria.

While, as yet, we have no proof of a specific causative virus, the nosologic independence of epidemic encephalitis appears proved when, following Stern, we assemble the main features: First, the clinical ones: (1) the prominence of somnolence, transient cranial nerve palsies, and atonic or hypertonic extrapyramidal motor disturbances (choreic and parkinsonian types); (2) the rarity of certain symptoms that are frequent in other forms of encephalitis, such as those indicative of destructive upper motor neuron lesions, as well as convulsions and anesthetics; (3) the relative frequency of "central" pains, sympathetic disturbances, especially salivation, and certain pseudospontaneous motions and disorders of respiratory rhythm, otherwise more commonly seen in psychogenic disorders than in organic disease; (4) the tendency to frequent variation in the grouping of symptoms in different epidemics and localities; (5) the presence of a chronic progressive form. To these we may add (6) the possibility of the development, after a free interval of years, of a progressive "para-encephalitic disease," analogous to the late nervous manifestations of syphilis. This usually is of a parkinsonian type, and may follow in the wake of an acute illness so mild that its nature becomes established only by the character of the subsequent symptoms.³

Anatomically, we have these principal features: (1) the insignificance of the gross changes; (2) the rarity of thrombosis and necrosis; (3) the lymphocytic character of the perivascular infiltrations; (4) the predilection of the inflammation for certain regions of the basal ganglions and brain stem.

Much work has been done on the spinal fluid, and the views, at first quite divergent, have become crystallized. There is no pathognomonic feature, but there are two fluid "syndromes," thus defined by Eskuchen:⁴ (1) lymphocytosis; globulin increase; luetic gold curve; increased sugar; (2) cell-globulin "dissociation," with relative increase in cells; luetic gold curve; increased sugar.

On the whole, the most valuable work in the last two years relates to the sequels, mental and physical, the number and variety of groupings of which are most surprising and bewildering, bringing new and unexpected problems in differential diagnosis. Postencephalitic states are easily confused not only with paralysis agitans and chronic choreas, but also with dementia praecox, general paresis, multiple sclerosis and brain tumor. Especially in children, in the absence of history, behavior disorders and other mental disturbances may lead to a wrong diagnosis of imbecility, "moral insanity," or plain unaccountable wickedness.—Editorial, *J. A. M. A.* (March 10) 1923.

3. This aspect of the encephalitis problem was discussed by Bassoe, Peter: *The Diagnosis of Epidemic Encephalitis*, *J. A. M. A.* **79**:2223 (Dec. 30) 1922.

4. Eskuchen, Karl: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **76**:568, 1922.

THE TEST OF HYPERTHYROIDISM

The limitations of the so-called Goetsch test as an index of hyperthyroidism have already been pointed out in *THE JOURNAL*.¹ This reaction consists in the response of patients to subcutaneous injections of small doses of epinephrin, a positive result being manifested by an increase in pulse rate and rise of blood pressure, exaggeration of tremor, palpitation and nervousness, appearing within a short time after the injection. The epinephrin was at first believed to produce sensitization of the organism so that a synergistic action of the thyroid secretion would manifest itself by pressor effects. Although Goetsch originally thought that the phenomenon might be of value in the diagnosis of thyroid disease by allowing a surplus output of thyroid hormone to manifest itself more readily, he later pointed out that the reaction, after all, is indicative only of alterations in the sympathetic system; that is, it becomes an index of sympathetic overstimulation.

In recent studies at the Department of Pharmacology at Columbia University, Lieb and Hyman² have demonstrated anew that the reaction to epinephrin measured in the response of blood pressure varies widely with the condition of the subject. This is contrary to the widespread belief that the circulatory system will respond with the accuracy of a chemical balance to any dose of epinephrin.³ These studies point to the frequency of "variables" in the involuntary nervous system not due to sensitization. They are frequently present when evidences of "tire" are present, and are definitely not associated with hyper-tonicity of the portion of the nervous system referred to.

Of foremost importance is the observation that the apparent "sensitization" by epinephrin may develop or persist even in the complete absence of thyroid glands. This fact at once indicates that the reaction is due to alterations in the peripheral structures of the involuntary nervous system rather than to changes in the thyroid. The demonstration that the involuntary nervous system is not a "constant" in its responses, and that the variations need not be due to sensitization by some artificially introduced variable such as the thyroid hormone, removes a fundamental postulate of the Goetsch test. It also jeopardizes the validity of the theory which holds that exophthalmic goiter is due to a synergism between the suprarenal medulla and the thyroid gland. As usual, the conflict of opinion is with theories of pathogenesis, not with the facts of direct observation.—*Editorial, J. A. M. A., March 24, 1923.*

1. The Goetsch Test in Healthy Persons, editorial, *J. A. M. A.* **79**:136 (July 8) 1922.

2. Lieb, C. C., and Hyman, H. T.: Studies of Graves' Syndrome and the Involuntary Nervous System. IV, The Vascular Response of the Pithed Cat to Single Intravenous Injections of Adrenalin, *Am. J. Physiol.* **63**:60 (Dec.) 1922; V, The Vascular Responses of the Pithed Cat to Repeated Intravenous Injections of Equal Doses of Adrenalin, *ibid.*, p. 68; VI, Attempts to Alter the Vascular Response of the Pithed Cat to Repeated Injections of Similar Doses of Adrenalin, *ibid.*, p. 83; VII, On the Mechanism of Sensitization to Subcutaneous Injections of Adrenalin, *ibid.*, p. 88.

3. Elliott: *J. Physiol.* **44**:374, 1912.

CORRECTION

In the article Recent Developments in Electrodiagnosis by Percival Bailey in the April issue of the ARCHIVES on page 441 the equations should read:

$$\frac{a}{b} = RC \left\{ \frac{I_o}{I_{rh}} - \log_e \frac{I_o}{I_{rh}} - 1 \right\}$$

$$\frac{a}{b} = RC (2 - \log_e 2 - 1) = 0.31 RC.$$

instead of as printed.

AMERICAN NEUROLOGICAL ASSOCIATION

The forty-ninth annual meeting of the American Neurological Association will be held in Boston, May 31, June 1 and 2, 1923.

AMERICAN PSYCHIATRIC ASSOCIATION

The annual meeting of the American Psychiatric Association will be held June 19 to 22, 1923, inclusive, at Hotel Statler, Detroit, Mich.

Abstracts from Current Literature

STUDY OF ANTIPROTEOLYTIC FERMENTS, AMYLASE AND OXYDASE IN FOUR TYPES OF PSYCHOSIS, WITH ESPECIAL CONSIDERATION OF METABOLISM IN MENTAL DISEASE. YOSCHIAKI HAYASCHI, *Monatsch. f. Psychiat. u. Neurol.* **51**:23 and 94 (Jan. and Feb.) 1922.

The author summarizes the results of his detailed studies: (1) Dementia paralytica: (a) In the acute stage or in an exacerbation of a chronic case the antitryptic content of the spinal fluid is increased. (b) The antitryptic power of the spinal fluid in the chronic stage is very variable; usually there is little or no increase. (c) There is some parallelism between the severity of the case and the antitryptic power of the spinal fluid. (d) The antitryptic power of the spinal fluid is greater in the acute than in the chronic phase. (e) There is some parallelism between the antitryptic power of the serum and that of the spinal fluid. (f) In some cases the amylolytic power of the serum is normal. (g) Remissions of the disease are accompanied by an increase, exacerbations by a decrease, in the amylolytic power of the serum. (h) The amylolytic power of the serum is reduced during the violent outbursts. (i) The amylolytic power of the spinal fluid is very variable; phases of violence and fear are accompanied by a reduction, remissions by an increase. (j) The quantity of oxidative ferments in the serum is generally above normal. (k) The quantity of oxidative ferments in the serum is greater in the violent phases than in the quiet periods. (l) It is probably greater in times of paralytic attacks than in the periods without attacks. (m) The quantity of the oxidative ferment in the spinal fluid is generally increased about 0.64.

(2) Dementia praecox: (a) In the acute phase, or in an exacerbation of a chronic case, there is usually a slight increase in the antitryptic content of the spinal fluid. (b) In chronic stages, the antitryptic power of the spinal fluid is nil. (c) In stuporous states, the antitryptic power of the spinal fluid is very variable. (d) In manic states, the antitryptic power of the spinal fluid is increased more than in stuporous states. (e) The antitryptic power of the spinal fluid is never higher than in progressive paralysis. (f) There is in general a parallelism between the antitryptic power of the serum and the spinal fluid. (g) The amylolytic power is normal. (h) The amylolytic power is never higher than in progressive paralysis. (i) The amylolytic power of the spinal fluid is very variable. (j) Excitement is associated with a decrease, quiet with an increase of the amylolytic power. (k) The oxidative ferments of the serum are generally above the average normal. They are reduced during excited phases, increased during quiet stages. (l) In periods of violence, the oxidative ferments of the serum are greatly increased. (m) In stuporous states the oxidative ferments of the serum are more increased than in the violent phase. (n) The oxidative ferments of the spinal fluid are increased above 0.6.

(3) Mania: (a) The antitryptic power of the serum varies within normal limits; that of the spinal fluid shows no change. (b) In many cases, the amylolytic power of the serum is normal; violent phases are accompanied by diminution, quiet stages with increase. (c) The amylolytic power of the spinal fluid

shows considerable variation. Excitement stages are accompanied by diminution, quiet phases by increase. (d) The oxidative ferments of the serum are below the average, and often below the low normal. (e) Exacerbation is associated with diminution of the oxidative ferment, remission with increase. (f) The oxidative ferment of the spinal fluid is in general below 0.6.

(4) Melancholia: (a) The antitryptic power of the serum varies within normal limits; it shows no variation in the spinal fluid. (b) In melancholia simplex the amylolytic power of the serum is normal. (c) In fear agitation the amylolytic power of the serum is reduced. (d) In fear agitation, the amylolytic power of the spinal fluid is also reduced. (e) The oxidative ferment is below the lower limit of normal. (f) The oxidative ferment content of the serum in fear agitation is above the average normal. (g) The oxidative ferment content of the spinal fluid is generally reduced below 0.6.

(5) Diagnostic notes: (a) It is noteworthy that the antitryptic power of the spinal fluid in dementia paralytica and dementia praecox is increased; in mania and melancholia is within normal limits. (b) There is no relationship between the strength of the amylolytic power of the serum and the type of psychosis, but it is interesting to note that remissions in progressive paralysis are associated with increase in the amylolytic power. (c) Contrary to Kafka's views, there is no connection between the strength of the amylase in the spinal fluid and the type of psychosis. (d) It is noteworthy that the oxidative ferments of the serum are generally above the average normal in dementia paralytica and dementia praecox, and below the average normal in mania and melancholia, with the exception of the fear agitations. (e) It is interesting to note that the oxidative ferments in the spinal fluid in dementia paralytica are over 0.64, in dementia praecox over 0.60, and in mania and melancholia reduced below 0.60. (f) If these data should be confirmed on a larger series of cases, it would be of importance in the differential diagnosis of these conditions.

At the conclusion of an elaborate discussion of metabolism, normal and abnormal, of nucleins, proteids and sugars, he elaborates the following hypotheses for the four psychoses: (1) In dementia paralytica: (a) The disturbance of protein metabolism is not the result of disturbance of the oxidative ferment but of disturbance of the proteolytic ferments and the de-aminizing ferments which hasten hydroclastic reaction and hydrolytic cleavage. (b) The disturbance of nuclein metabolism is the result of disturbance of a true oxidative ferment—xanthoxydase. (c) The disturbance of oxidation of carbohydrates can be explained on the basis of an incomplete action of carboxylase which hastens the Cannizzaro reaction. (2) In dementia praecox: (a) The disturbance of protein metabolism is not caused by disturbance of actual oxidative ferments but by disturbance of proteolytic ferments. (b) The disturbance of nuclein metabolism is caused by disturbance of actual oxidative ferments, more particularly xanthoxydase. (3) In mania: (a) Catabolism and oxidation are increased. The cause of this is unknown. (b) Oxidation processes are disturbed because of lack of oxidative ferments, in spite of increased oxidation, and for this reason run in false tracks. (4) In melancholia simplex: Oxidation processes are not disturbed but catabolism and oxidation are slowed. What causes this is unknown. (5) In involution melancholia: The disturbance in oxidation is not caused by disturbance of the actual oxidative ferment but through a great need for oxygen, or through a disturbance of the fermentative processes which produce the hydroclastic reaction and hasten the related Cannizzaro reaction.

SELLING, Portland, Oregon.

HISTOPATHOLOGIC STUDIES IN PSYCHIATRY. W. SPIELMEYER, *Klin. Wehnschr.* 1:1817 (Sept. 9) 1922.

In this article the author reviews some of the fundamental facts in regard to the histopathology of the cortex. He asserts that it is disappointing in a study of such striking conditions as confusional states and epilepsy to find so little of positive value; and if we should review our knowledge of the anatomy of mental diseases, very few conditions could be named in which a definite anatomic lesion could be pointed out. Syphilitic and arteriosclerotic conditions, senile changes, several types of idiocy and a few other isolated conditions make up the entire group about which we have any definite knowledge.

It is not necessary to preach about the hopelessness of anatomic study in psychiatry; the lack of progress in its development is merely a seeming standstill, and one familiar with this field of work cannot be surprised at this when he remembers how little is known about the more commonly recognized cortical conditions. Thus far it is only the gross changes which help us, rarely ever nerve tissue changes. Even in general paralysis and arteriosclerosis the alterations which we study occur in the mesenchyma.

In the large group of degenerative diseases but few histopathologic changes are of diagnostic value. The plaques in senile dementia, the ganglion cell changes in Alzheimer's disease and in amaurotic idiocy are outstanding exceptions. Even in dementia praecox in which one would expect to find changes following so much study, investigations up to the present time have been very unsatisfactory.

If one recalls how difficult it is to recognize the ganglion cell changes in the motor groups in such profound conditions as spinal muscular atrophy or bulbar palsy, or to recognize a loss of ganglion cells in idiopathic muscular atrophy, one need not wonder at the difficulty in discovering changes in cells and fibers in the cortex in conditions without disturbance of functional activity. The same difficulty in determining an exact qualitative change is also to be noted in neurologic conditions such as tabes and amyotrophic lateral sclerosis in which the pathologic diagnosis is not based on the character of the nervous tissue change, but on the recurrence of a similar and localized process. Spielmeyer doubts if similar, closely circumscribed, system diseases of the cortex occur. Certainly they are so rare that they have no practical value.

It is interesting that the end results of various processes, as also noted in other organs, resemble one another very closely, and indicates the need of study of the earlier stages of disease. In the past ten years the study of the ganglion cells has again come into its own, and we no longer look for changes in the nerve fibers but study the changes in the nerve cells. To meet this difficult problem, histochemical methods offer a field which is approached with new hope. The study of the neuroglia is of considerable importance. Its reaction is independent of the quality, the grade and the tempo of the disease process. Just how the glia cells dispose of waste products, if they function as phagocytes or if they are affected by the disease process, is of extreme importance, as also the method of protoplasmatic and added changes in the parenchyma.

As Spielmeyer has pointed out in his "*Histopathologie des Nervensystems*" considerable advance has been made, even though an anatomic reason may not have been determined as yet for isolated disease entities. As the cortical structure is so complicated, attention should be directed to the more simple parts

of the nervous system; the cerebellum has shown changes in chronic processes, as also the Ammon's horn in epilepsy. Anatomic changes should not be considered conclusive until proved by repeated examinations and substantiated clinically.

Alzheimer, with the clinical assistance of Kraepelin, in a most painstaking manner, worked out the histopathology of the disease named after him. The work on amaurotic idiocy has been checked sufficiently to establish a definite picture. The same is true of Wilson's disease and pseudosclerosis. Isolated anatomic findings are of importance, as, with correlation of similar cases, disease groups may be established. This has been demonstrated so well by the diligent and elaborate work of Nissl. Today clinics and well regulated laboratories that work up their material from the clinical and pathologic standpoint can hope to make progress. It is true that pathologic work is chiefly interested in differential diagnosis as in dealing with general paralysis, cerebral syphilis, arteriosclerotic changes and the like. Spielmeyer has seen a case of multiple sclerosis with marked cerebellar changes considered as general paralysis and only correctly diagnosed by microscopic study. The same may be said of Alzheimer's disease and other senile conditions. General paralysis has been taken for dementia praecox and only on section has the error been discovered. Much work of merit has been done on amaurotic idiocy, tuberous sclerosis, and also on traumatic, circulatory, encephalitic, and syphilitic conditions. The study of general paralysis has become more interesting of late due to the work of Jahnke. It is possible that the question of specificity of the spirochete for the nervous system may be answered, as also its reaction on the nervous tissue and blood vessels. Among the encephalitides, general paralysis has the interesting finding that besides the inflammatory changes, there are also associated degenerative changes. Such secondary degenerative changes also occur in spotted fever and epidemic encephalitis. The histologic problems in diagnosing diseases are not settled by indicating the localization of the process and its characteristics. Everybody knows how difficult it is to make a complete and careful histologic study of the brain. The entire problem of histopathology is still in its developmental period and many fundamental facts are as yet unsolved. Thus we know nothing as to why such definite processes as general paralysis and senile dementia affect the Ammon's horn and frontal lobes and why the occipital lobes are left more or less unaffected. The frequency with which the third layer of Brodmann is affected by various diseases also remains a curiosity. We have arrived at no definite conclusion, nor do we know as yet what local pathogenic factors are at work causing some processes to prefer certain cortical zones, and, as Spielmeyer emphasizes, there still remains a great deal of work to be done in the field of histopathology.

MOERSCH, Rochester, Minn.

THE ASSOCIATION BETWEEN TUBERCULOSIS, NEUROPSYCHOPATHY AND DELINQUENCY. ROSOLINO COLELLA, Riv. d. Patol. nerv. e ment. 27: Oct. 15, 1922.

The problem must have special significance in Sicily where the author is director of the department of nervous diseases in the Universities of Messina and Palermo and also of the Hospital for the Insane of the latter city, which contains about three thousand patients. In his clinic and hospital population, 40 per cent. had tuberculosis, active or latent, and 10 per cent. had syphilis. It was noted by the author alone in 1894, and in conjunction with Frisco in 1914,

that tuberculosis was encountered very frequently in the nervous system. They called attention not only to tuberculous meningitis, tuberculosis of the brain, myelitis, and areas of softening due to thrombosis caused by changes in the vessel walls or to embolism, but also to certain psychic, motor, sensory and trophic disorders. The author also has described tuberculous polyneuritis with or without psychosis. He again describes pathologic and clinical examples of all these conditions, and pays particular attention to the antisocial and delinquent tendencies of those suffering from tuberculosis. The author describes in detail five cases, three of which were subjected to exhaustive anatomic studies. The author investigated spinal nerves, roots and the ganglions of the cord, medulla and pons, the cranial nerves, various portions of the cortex and various muscles. In all cases, necropsies were performed within twenty-four hours. The methods of staining used were those of Orth, Hoyer, Nissl, Weigert and Golgi. He also used methylene blue and hematoxylin. Parenchymatous changes were found in every part of the peripheral nerves, motor, sensory, and mixed, spinal and cranial. These changes were equally as marked in the central nervous system and the spinal roots as in the peripheral nerves. They resulted in either severe motor, with predominant trophic, disturbances or in special painful or anesthetic neuritides. Psychic disturbances were associated with multiple neuritis. Polyneuritis was the first sign in an otherwise latent case of intestinal tuberculosis. Of all the disturbances caused by the tuberculous toxins in the cerebrospinal axis, the mental disturbances presented the most varied picture. These the author differentiates from the polyneuritic psychosis. To the causes of this type of psychosis, namely, influenza, puerperal fever, erysipelas, diabetes, acute rheumatic fever, smallpox, typhoid pneumonia, syphilis, alcohol, lead, arsenic, etc., the author long ago added tuberculosis.

This picture is quite definite and has been described by many authors. But in three of the author's cases there were syndromes of pseudoparesis, manic depressive insanity and dementia. In these cases were found not only degeneration of the peripheral nerves, muscular atrophy, changes in the columns of Goll, vacuolization of the ganglion cells of the anterior horns, softening of the superficial portions of the cortex, but also colloid degeneration of the thyroid. This last finding would point to a toxemia.

The author calls attention especially to the changes in the association fibers of the brain to explain the special characteristics of the polyneuritic psychosis. That the other psychotic symptoms are varied can be seen by the stress laid on various types observed in the experience of many writers. Thus Marie noted the hypochondria, Berenberg the apathy, Ball the unsystematized delirium, Peter the neurasthenia, Boccilli the moral perversions, Clouston the mania of suspicion, etc. The symptoms in the individual case, therefore, vary from simple loss of memory, to impulsive behavior, epileptic seizures with or without psychosis, hallucinations, confusions and occasionally even complete dementia. These symptoms are often preceded by a period of anxiety and great irritability. Occasionally, the first sign of mental trouble is delinquency or immorality. The author makes a great point of his conception that "regardless of the fact that tuberculosis is the causative factor in these cases of nervous and mental disease, the subjects must have a predisposition to neuropathic and psychopathic disturbances." Of his five cases, there was insanity in the mother of the first case, hysteria and alcoholism in that of the second, alcoholism and insanity in the father of the third, etc.

Three of the author's patients committed grave antisocial acts and the author recalls many horrible murders committed in Italy by tuberculous patients. One

killed his sister in the hospital, another his physician, still another the leader of a charitable organization who had assisted him, etc. The author recalls the experiments of Neircoli in which perfectly normal, intelligent, affable patients with tuberculosis became psychically much disturbed after injections of tuberculin.

In the author's experience some of the most common antisocial acts are of a sexual nature. Perhaps even more common than this type of delinquency are the acts against the person. The author thinks that the nucleinic acid contained in the tuberculosis poison, and also the principal component of purin, causes these violent acts by reason of its power to excite function, etc. The crimes against the person committed by the author's patients were committed in a perfectly insane, absurdly brutal, impulsive manner. The author recites in detail, the case of the fifth patient who killed his own wife. In this case the author was able to satisfy the judges that the man was insane, as a result of his tuberculosis. He was committed to a hospital for the insane.

The author also makes a point of the fact that in his experience not only the mental but also the polyneuritic symptoms often improve correspondingly with the improvement in the tuberculosis of the lungs or elsewhere.

In the discussion which followed the reading of the author's paper, it was brought out that in none of the author's work, was he ever able to find the tubercle bacillus, either in the peripheral nerves or in the central nervous system. It was also brought out that he did not feel that there was anything particularly characteristic about the types of mental reaction seen in tuberculosis; perhaps the most frequent reactions were those that resembled paresis, dementia praecox and manic depressive insanity. This, of course, does not take into consideration the typical Korsakow reactions in polyneuritis.

OSNATO, New York.

HYSTERICAL GANGRENE. G. C. BOLTON. *Monatsch. f. Psychiat. u. Neurol.* **51:1**, 1922.

Numerous vasomotor and trophic disturbances have been described in hysteria: erythema, pemphigus, herpes, purpura, alopecia, premature canities, loss of healthy teeth. There is also hysterical edema, usually associated with hysterical paralysis and contractures, but sometimes independent of them. Charcot described a special form to which he gave the name "Oedème bleu des hystériques." He proved its hysterical origin, in that he could produce it by suggestion under hypnosis, and remove it by suggestion. The type which he described is simply one of many forms of trophoneurotic edema, all variants of a single trophic disturbance. They occur not only in hysterical patients, but also in individuals with neuropathic constitutions. Closely related to them is hysterical gangrene.

Hysterical gangrene has given rise to much disagreement. Some admit its existence, others, with Babinski feel that it is always an artefact (mutilations). Many of the cases are undoubtedly artefacts, many cases of swelling and edema are due to the application of bands, and many of skin necrosis are due to the use of irritants. Careful observation of the patient in bed will allow one to determine definitely whether or not artificial means have been used to produce the lesions. Hysterical gangrene may occur in three forms: (1) Solitary hysterical gangrene. (2) Multiple neurotic gangrene. (3) Herpes gangrenosus hystericus.

Herpes gangrenosus hystericus appears as a herpetic lesion, but differs from ordinary herpes in that it does not conform to a skin nerve or root zone. After the vesicles break, the epidermis desquamates and does not regenerate; in its place appears a superficial patch of gangrene, usually surmounted by a crust. The disease is very obstinate, and resists treatment. If the superficial necroses are artificially produced, they are larger, more irregularly distributed, and heal readily under appropriate treatment.

In multiple neurotic skin gangrene, a number of superficial necroses develop, usually after trauma; the first patch usually appears at the site of the trauma; the interval between the trauma and the onset of gangrene varies from hours to months. The patches are anesthetic; they are often symmetrically placed; in severe cases they may involve almost the entire body. Some cases heal readily, others are very refractory. All these patients are neuropaths, many of them severe hysterics.

Solitary hysterical skin gangrene is much less common than the other types. The gangrenous patch goes deeper and involves all layers of the skin. The author reports a case of this type, in which all chance of artefact was excluded. The patient was 25 years old, unmarried, and very nervous. She belonged rather to the group of constitutional psychopaths than to true hysteria. She was a prostitute and was sent to the hospital to be examined for venereal disease. None was found. While in the hospital she broke a small vase, and in so doing received a slight, superficial wound on the palmar surface of the right hand. A small splinter of glass stuck in the skin; it was easily removed with a forceps. The wound was not infected. This glass splinter worried the patient and she believed that her hand would have to be amputated. Shortly after the injury, there appeared on the back of the hand an area of edema, circumscribed, elastic and rather firm, which the author considers hysterical. The edema disappeared after five days. The zone about the wound on the palm began to show changes on the second day—first anaemic, then brownish, and on the third day, black. This developed into a typical area of gangrene, which included the whole zone of the original wound, and involved skin and subcutaneous tissues. After twelve days the scab began to loosen, and complete healing took place.

In the history of the patient, there are numerous evidences of disturbance in the vegetative nervous system—paraesthesias in hands and feet, dead fingers, feeling of oppression, hyperacidity, dysmenorrhoea, and attacks of urticaria. She also showed such vegetative stigmata as dermatographia, sinus arrhythmia, and oculocardiac reflex.

As to the mechanism of production of these lesions, the author offers little. He points out that what one can suggest to a patient in hypnosis, the patient can suggest to himself. And whereas hysterical gangrene has never been definitely produced by hypnotic suggestion, closely allied forms of edema have been so produced and cured. He agrees with Lewandowsky that this entire group of disturbances cannot strictly be classed as hysterical. They occur not only in hysteria, but, as in the above case, in other psychopathic states as well. He believes that a functional vasomotor insufficiency, congenital in origin, is the main factor—a constitutional sympathetic insufficiency, which in turn depends on an insufficiency of those endocrines which keep up the tone of the sympathetic system, especially the thyroid and the chromaffin system.

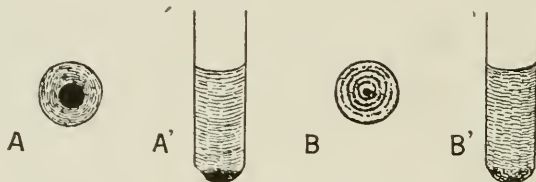
SELLING, Portland, Oregon.

THE BENZOIN COLLOIDAL REACTION IN 105 NEUROLOGIC CASES.

P. MARIE, BOULTIER and IORGULESCO, *Ann. de méd.* **12**:263 (Oct.) 1922.

The writers recall that this reaction rests on the mixing of a certain volume of a colloidal suspension of benzoïn with an equal volume of cerebrospinal fluid diluted with water in proportions of 3:4, 1:2, 1:4, 1:8, 1:16, 1:32, etc. The phenomenon consists of precipitation of benzoïn in the case of specificity. The two ways of doing the test, with five tubes, "reaction simplified," or with sixteen tubes, "reaction complete," have given identical results as regards diagnostic value.

They believe that certain directions are important for the reading of the tests. Often, both in a tube of negative reaction, and in a tube of positive reaction, or, even more, in the case of a subpositive reaction, the mass of the liquid in the tube shows approximately the same clearness. It then becomes necessary to examine the base of the tube. A precipitation due to simple decantation, and indicative of a negative test, can be seen to differ in arrangement from that of a precipitation determined by flocculation (positive). In the negative reactions there is a compact round mass, sharply delimited, situated in the very center of the bottom of the tube. In contrast, in the positive reactions, the bottom of the tube does not present a central pin point mass. Instead there are several concentric rings of precipitate. The diagrams make this clearer.



Precipitate: A and A', base and side of tube in a negative reaction; B and B', base and side of tube in a positive reaction.

Various authors affirm, with reason, that it is possible to read the positivity after 15 minutes and recommend a definite rereading at the end of twelve hours. However, as some positives are late in evolving, the present writers recommend that the second reading be done twenty-four to thirty-six hours after the first. The test proved positive in 33 cases. It was negative in 4 cases of brain tumor, 12 cases of epidemic encephalitis, 1 case of Pott's disease, 6 cases of epilepsy, 2 of psychosis, 1 of senility, and in 56 cases of diverse character, including many functional disorders. The test gave reactions which were parallel with the results of Bordet-Wassermann reactions in all but three cases (two hemiplegias and one Erb's paraplegia), and, in these, later events favored the result obtained with the colloidal benzoïn test.

DAVIS, New York.

THE REACTION OF COLLOIDAL BENZOIN IN GENERAL PARALYSIS. R. TARGOWLA, *Presse méd.* **30**:931 (Oct. 28) 1922.

The colloidal benzoïn reaction of Guillain, Laroche, and Léchelle uses 15 tubes of cerebrospinal fluid in dilutions from 3 in 4 to 1 in 16384. 22222.22222-10000 is the average reaction in general paralysis; "2" indicates total precipitation, "1" and "0", partial and absent precipitation. A "positive reaction" requires

total precipitation in the first five tubes, the "syphilitic zone." Partial precipitation in the first tubes, such as 12220.2222100000, or 11100.2220000000, is rare and indefinite. However, partial precipitation at the end of the "syphilitic zone" is frequently found: 22221. appears almost as diagnostic of general paralysis as does 22222.; and 22200. and 22100. are sometimes found in early cases of the disease.

The writer does not claim that his "positive reaction" is absolutely pathognomonic of general paralysis, but states that every positive benzoin reaction in a series of 400 spinal fluids was verified by typical clinical findings or by necropsy. Six cases in this series were clinically not general paralytics at the time the fluid was first taken, but developed typical symptoms later and thus verified the serologic diagnosis. During clinical remissions, the benzoin reaction remains positive at least to some extent, though less strongly so than during an active phase. Furthermore, the strength of this reaction varies directly with the organic neurologic element causing a period of exacerbation. For example, a strongly positive reaction during an episode of agitation, accompanied or not by an excess of urea content or hypertension of the spinal fluid, points toward an intense and rapidly progressive meningo-encephalitis, against which therapy will be useless; while if the benzoin reaction be only moderately or weakly positive, with excessive urea content and possibly hypertension of the fluid, the agitation is due to a toxic process and should be readily curable. Similarly, a weakly or moderately positive benzoin reaction with normal urea in the fluid during a period of excitement in a general paralytic, favors the diagnosis of a transient manic state.

HUDDLESON, New York.

EXPERIMENTAL OBSERVATIONS ON THE LOCALIZATION OF THE PAIN SENSE IN THE PARIETAL AND DIAPHRAGMATIC PERITONEUM. JOSEPH A. CAPPS and GEORGE H. COLEMAN, Arch. Int. Med. **30**:778 (Dec.) 1922.

The method employed in this study consists essentially of inserting a trocar through a previously anesthetized portion of the abdominal wall and passing silver wires, one beaded and smooth, another relatively sharp, and palpating various areas of the parietal and diaphragmatic peritoneum. In the earlier experiments patients with ascites were chosen; in the later series the abdomen was first inflated with air. It was found that the anterior and lateral portions of the parietal peritoneum, as far as explored, were sensitive to pain from strong pressure of a smooth point or light pressure or lateral movement of a rough point, and that this pain was localized by the patient with considerable accuracy, the error being less than one inch. The localization of pain from stimulation of the diaphragmatic peritoneum was found in no instance to be in the diaphragm but to be referred to some distant point. Stimulation of the outer margin gave rise to pain in the lower costal region and the sub-costal abdominal wall; stimulation of the central portion produced pain over a point along the ridge of the trapezius. The authors note that, embryologically, a portion of the diaphragm is derived from cervical myotomes which carry down in their descent portions of the third, fourth, and fifth cervical nerves; these nerves, originally both sensory and motor, fuse to form the phrenic nerve. The sensory function of the phrenic, however, is lost to the extent that while it transmits afferent stimuli, it is unable to register localized sensations in the diaphragm; these are referred along the course of the cutaneous branches of the fourth cervical segment.

VONDERAHE, Philadelphia.

PERUVIAN FOLK LORE. T. MEJÍA XESSPE. *Rev. de Psiquiat.* 4:205 (July) 1922.

In the first instalment of this paper, an attempt is made to analyze certain elements underlying Peruvian habits and beliefs. In the face of modern civilization the natives have retained their inborn melancholy, traditions and superstitions. They are averse to culture, medicine, hygiene and science in general, as they consider these only as masked ways of increasing their needs. As they live from hand to mouth, thrift and prevention are unknown. Life at home turns around its head, the father. Men, women and children spend the day out, the house being only a resting place and warehouse, or rather a "corral" where poultry, rabbits, cattle and humans dwell together. Work is divided among the family; while the father plows, the mother minds the cattle and the children gather wood. This monotonous life never changes except on such occasions as planting and harvesting time, housebuilding, christenings, weddings, burials and holidays. At sowing time, the owner after providing plenty of "chicha" at home, invites all his friends to join. Some mystic rites are indulged in, such as sprinkling the seed with "chicha," singing victory songs, throwing dirt at each other, laying aside the largest grains as amulets, and paying homage to the owners on completion of building. When a child is born, salt and water are placed in his mouth and the devil is warned to keep away. The first haircutting is turned into a celebration. Engagements are made at the age of 10 to 15, and marriages at 15 to 20, the whole matter being arranged by the parents. The best man must make a present of a house, some cattle and a sum of money; his example is followed by parents, relatives and guests. After the wedding dance is over, the newly married couple are locked in together until the best man opens the door during the next day. Sometimes the couple is tied together for a short time with a silver or gold chain or a wool cord as a reminder that they must not separate during life. Adultery is rare, and guilty parties are visited with contempt. Sex ignorance is so deep, that sometimes the bride, after first submitting to the husband's embraces, runs away in horror and even commits suicide. When a corpse is buried, flowers, coca and other herbs are thrown in to keep the dead man from rising, with chicha and water to keep him from burning in hell. After the burial, aromatic herbs and pieces of horn are also placed in as a protection against further deaths. If, on the way to the burial place, the remains seem heavy, the funeral is postponed, as this is considered evidence that the deceased is not yet willing to be buried. Single people and adulterers are buried with downturned faces, symbolizing their unworthiness to see God. In all cases, a few large stones are put on the body to prevent resuscitation.

MOLL, Chicago.

SUGAR IN THE BLOOD AND SPINAL FLUID. M. POLONOVSKI and E. DUHOT, *Presse méd.* 31:60 (Jan. 20) 1923.

This is an analysis of findings obtained by the examination of blood and cerebrospinal fluid taken practically simultaneously, in normal and in pathologic cases. A definite relationship is claimed between the sugar contents of the two fluids in normal individuals. Variations in this ratio are considered more valuable, for the study of pathologic cases, than variations in the spinal fluid sugar content alone. The normal sugar content of spinal fluid appeared to vary over a wider range than is usually believed. It was found to be 0.45 to 0.85 nine to ten hours after a meal, and 0.55 to 1.05 three to five hours after eating.

A reversible reaction seemed to obtain between free sugar and available sugar—explained on the basis of a physical rather than a chemical difference between the two—part of the sugar behaving like a colloid. Glycolysis, breaking down of sugar, was partly counterbalanced by an increase in directly measurable sugar, a "liberation" of sugar. Variable factors like these, hitherto disregarded or inadequately recognized, were deemed responsible for the divergent findings of various authors. Questions of technic as affected by the different colloidal properties of the two mediums (blood and fluid) are discussed in detail.

An equilibrium was noted between the free glucose of whole blood, and that of the spinal fluid, the latter being consistently a little lower. This equilibrium persisted under experimental conditions of hypoglycemia and hyperglycemia, e. g., as influenced by adrenalin; similarly, in diabetes. The increased sugar content of the cerebrospinal fluid commonly encountered in epidemic encephalitis is paralleled by an increase of blood sugar, and is therefore not a purely local phenomenon. In cases of meningitis with diminished spinal fluid sugar, the blood sugar must also be taken; if there is a disproportion between the two findings, its extent will be of diagnostic and prognostic value.

HUDDLESON, New York.

SPINAL ACTINOMYCOSIS. LESNÉ and BELLOIR, *Ann. de méd.* **12**:329 (Oct.) 1922.

According to Poncet 3 per cent. of cases of actinomycosis show central nervous system localization. In 1911, Arnaudon brought together twenty-seven observations of cerebral localization. In 1919, Sagredo reported two more cases.

Cerebral localization sometimes spreads from a buccopharyngeal lesion. Other times, it is secondary to an abdominal or pulmonary actinomycosis which reaches the brain by the blood stream. "Metastatic actinomycosis most often is due to emboli from the lung, for the lung is very frequently involved." Sometimes the cerebral lesion appears to be primary, with the portal of entry unknown (Enriques and Sicard). The symptoms are those of brain abscess or tumor. A fatal issue in coma is the rule. Occasionally, due to the rupture of an actinomycotic abscess, an acute meningitis ensues and in these instances, lumbar puncture permits one to find the parasite in mycelial tufts in the spinal fluid.

In several cases of vertebral actinomycosis, the spinal cord has been compressed, with resultant paraplegia or quadriplegia. Because the writers do not know of any case in medical literature of actinomycosis with definite location in the spinal cord tissue, they report, with necropsy findings, a case recently encountered. There were dual subcutaneous abscesses, severe and diffuse pulmonary disease with mycelia in the sputum, and in the third month, in spite of treatment, a rapidly established flaccid paraplegia with sphincter disturbance, and in three days total anesthesia in all modes over the legs and up to the level of the umbilicus. The spinal fluid showed only slight albuminosis and 8 lymphocytes per cubic millimeter. The serum of the patient agglutinated cultures of sporotricha and gave the fixation reaction of Vidal and Abrami.

Necropsy showed a grouping of very small abscesses in the lower dorsal portion of the cord, each no larger than a millet seed. These were conglomerate inflammatory nodules, almost all of them having typical yellow actinomycotic granules in the center. Near the center of the cord there was dense conjunctivovascular tissue, and more peripherally, and between the small abscess points, intense neuroglial thickening and fibrovascular spaces.

Iodid treatment in actinomycosis is said to be both curative and preventive of emboli. But this form of treatment, while usually so active in cutaneous mycosis, appears to be without any effect in the mycosis of the central nervous system. Finally, in actinomycosis it is well to avoid surgical intervention as much as possible for it can frequently instigate metastasis and generalization.

DAVIS, New York.

OCCIPITAL LOBE EMBOLISM. ANDREW H. WOODS, *J. Nerv. & Ment. Dis.* **55:2** (Feb.) 1922.

The first patient, a woman, aged 30 years, developed pain and stiffness in the left side of the neck. After two treatments by an osteopath, right homonymous hemianopia with paresthesia and weakness of the left limbs developed; the latter cleared up within a week. Examination three months later revealed a right homonymous upper quadrantanopia with involvement of one fourth of the macular field. Pressure on the tip of the first and second transverse cervical processes produced pain and increased resistance in the soft tissues of the left suboccipital region, behind the mastoid process. Bending to the right and rotation of the head also caused pain in this region. Laboratory reports and neurologic examination were negative.

The second patient, a man, aged 55 years, was seized suddenly with dizziness, momentary tingling in the left extremities, left homonymous hemianopia, visual paresthesia and disorientation. Two weeks later the visual fields had partly cleared up in the peripheral zone and in thin sectors in both upper and lower quadrants near the vertical meridian. There were no abnormal neurologic findings, but valvular heart disease was present.

The author regards the internal carotids or vertebral arteries as the commonest route of the emboli and states that a backward current through the posterior communicating artery may sweep an embolus from the carotid into the posterior cerebral artery. He found no similar case in the literature. The first embolism is believed to have come from the left vertebral artery and to have been caused by a suppurative process in the surrounding cervical tissues. The immediate mechanism of the tract irritation may have been disturbance of the local blood supply of the cervical cord or merely mechanical.

In the second case the embolus is believed to have affected the sensory tracts in the right thalamic region, and thus to have produced the paresthesia, since this region is supplied by the posterior cerebral artery. From a study of the visual fields, the author believes that the right half of each macula sends fibers only to the right occipital cortex, and the left half only to the left occipital cortex. Partial recovery, such as was seen in the retinal fields in the two cases described, may be due, in the author's opinion, to a slight collateral blood supply, particularly in areas at the periphery, or to a shrinkage of the thrombus. There may have been an initial transitory depression of function in the surrounding areas due to concussion.

STACK, Philadelphia.

THE DIAGNOSIS OF SILENT INTRACRANIAL ABSCESS IN THE AMBULATORY STAGE. HENRI ABOULKER, *Presse méd.* **30:474** (June 3) 1922.

The thesis of this paper is that patients with brain abscesses are typically ambulatory up to the precomatose stage; but that the lesions could be dis-

covered and operated on earlier than is now usual. They should be recognized while the patient is still working. The two cardinal symptoms then, as later, are headache and clouding of the intellect with psychic depression. Differentiation must be made from migraine and neurasthenia.

A number of cases are reported, leading to these additional conclusions: (1) that large extradural abscesses, frontal, temporoparietal, or even cerebellar, may remain strictly silent; (2) that the classical symptomatology of encephalic abscess is frequently absent, even in extensive destruction of the temporo-sphenoidal lobe or of the cerebellum (this symptomatology is ascribed to an increase of pressure in an aseptic cerebrospinal fluid which may develop without an abscess and may or may not occur with one); (3) that local anesthesia should be used at operation, chloroform especially being avoided; (4) that motor disorders are not suggestive of brain abscess, at least not of abscess of otitic origin, since the causes of such cerebral symptoms of a motor character as occurred in the series studied, were found to be quite independent of otitis or abscess.

HUDDESON, New York.

STUDIES IN ASYMPTOMATIC NEUROSYPHILIS. III. THE APPARENT INFLUENCE OF PREGNANCY ON THE INCIDENCE OF NEUROSYPHILIS IN WOMEN. JOSEPH EARL MOORE, Arch. Int. Med. **30**:548 (Nov.) 1922.

Moore finds in his group of cases that clinical neurosyphilis is three times as frequent in men as in women, although the nervous systems of both, as indicated by spinal fluid findings, are invaded with equal frequency in the early stages. Early syphilis, the author notes, is markedly altered by the simultaneous occurrence of pregnancy, when grave syphilitic lesions tend to be inhibited; spinal fluid abnormalities indicative of syphilis are twice as frequent in a group of sterile women as in a group in which one or more pregnancies have occurred since infection. In consideration of the above data, the author suggests that pregnancy is one factor which may account for the comparative freedom of women from neurosyphilis.

VONDERAHE, Philadelphia.

IS THE STOMACH A FOCUS OF INFECTION? NICHOLAS KOPELOFF, PH.D., Am. J. M. Sc. **165**:120 (Jan.) 1923.

Kopeloff presents a further account and résumé of chemical studies in mental disease and attempts to answer the following question: Has Rehfuß demonstrated that the stomach is a seat of focal infection? Psychiatrists should be highly interested in the report in view of Cotton's claims that: "The stomach and duodenum are very frequently the seat of secondary foci. . . . The bacteria invade the stomach wall and appear to interfere with the secretion of hydrochloric acid, so necessary to digestion. Cultures of the stomach contents will reveal the presence of various types of streptococci and frequently of various types of colon bacilli. The chemical examination of the stomach contents will show either a very low secretion of hydrochloric acid, or, in many cases, its entire absence during the test meal."

The author's chief objections to the method of Rehfuß are: first, that there is little or no value in single determinations; and, second, that he (Rehfuß) neglected consideration of the ingested saliva, and that thereby a tremendous margin of error was produced.

The following observation contains the keynote of Kopeloff's findings: "Bacterial counts showed a striking reduction in numbers in gastric fractions when saliva was inaccessible. The highest number of bacteria per cubic centimeter in a psychotic patient (manic-depressive: manic state) where saliva was *not* removed was 48,000; where saliva *was* removed the highest number found was 32. Similar results were obtained with a normal individual and with other manic patients." The significance of these statements is enhanced when it is recalled that it is a matter of great difficulty to prevent the swallowing of saliva by disturbed and excited patients, and the reduction in the number of bacteria occurred irrespective of the degree of gastric acidity. No normal bacterial flora has been demonstrated; the yeasts, staphylococci, streptococci, and members of the lactic-acid and aerogenes groups which were recovered from the stomach were also found either in the saliva of the same patient or in food which had been given. The conclusions: "The bacteria content of the stomach is influenced by the saliva. The Rehfuess method of fractional gastric analysis cannot be considered an adequate criterion in determining whether the stomach is a focus of infection," seem justifiable and well within the limits of the premises outlined in this article.

STRECKER, Philadelphia.

TREATMENT OF GENERAL PARALYSIS WITH RELAPSING FEVER VIRUS. S. LORENTE Y PATRÓN, *Rev. de Psiquiat* **4**:193 (July) 1922.

Six male patients with general paralysis were treated with relapsing fever virus, according to Rosenblum's method, and to recent suggestions by Mülhens, Weygandt, Kirschbaum, Plaut and Steiner on the advantages of this procedure. In three subjects, fresh defibrinated blood from a relapsing fever case was used; in one, the inoculation was made directly at the bedside of the relapsing fever case; and in the other two, the blood was taken from the general paralytic who had been inoculated directly, after the presence of the *Spirochaeta recurrentis* has been verified. These last two proved absolutely refractory to the relapsing fever infection, the virus having apparently lost its potency in the transmission. The four successfully inoculated patients (aged 57, 57, 44 and 38 years) improved in varied degrees, especially as regards clinical signs and weight. The cerebrospinal tests, except the colloidal benzoin test, showed little change. Only one 3 c.c. dose of blood was used, except in one case, in which 5 c.c. were injected. Two of the patients had previously been inoculated with malarial blood. The injections were invariably followed by four paroxysms. The patient who presented most clinical improvement was the one who received 5 c.c. of blood, although previous malarial virus injections had yielded no results at all. As these cases were treated between February and May, 1922, and were reported before July, 1922, no definite conclusions can be drawn as to the value of the treatment.

MOLL, Chicago.

ON THE VISCOSITY OF THE CEREBROSPINAL FLUID. T. SODA, *J. Nerv. & Ment. Dis.* **54**:227 (Sept.) 1921.

The viscosity of the cerebrospinal fluid was determined in twenty-seven cases of disease. In addition, the following constituents of each fluid were determined: protein, total nitrogen, chlorid, inorganic phosphate, alkalinity, and the number of cells. The viscosity showed a tendency to increase with increasing amounts of protein; there was also a parallel between the curves

of alkalinity and viscosity, and between the number of cells and viscosity. The other constituents studied played only a minor part. The author concludes that viscosity determinations of the cerebrospinal fluid are of doubtful value.

VONDERAHE, Philadelphia.

AN UNUSUAL CASE OF MORPHINE POISONING. LEYSER, *Monatsch. f. Psychiat. u. Neurol.* **51**:12 (Jan.) 1922.

The author reports the case of a morphine addict who developed an acute mental upset (a delirious dream-state), accompanied by certain physical abnormalities: slurring speech; tremulous writing; absent pupillary reflexes; nystagmus; volitional tremor; past pointing; tendency to fall to the left; absent abdominal reflexes. Complete recovery occurred in six days. The differential diagnosis is discussed and the author concludes that the case is one of acute morphine poisoning, although no similar case is on record in the literature. The general picture resembles Wernicke's acute hemorrhagic polioencephalitis. Similar cases due to alcohol, veronal and botulism have been reported. In support of his theory, the author calls attention to the similarity between the action of alcohol (definitely known to produce such syndromes), and morphine.

SELLING, Portland, Oregon.

AN EPIDEMIC OF BOTULISM. C. MOREL and R. DE ST. MARTIN, *Presse méd.* **30**:829 (Sept. 27) 1922.

One tin of American canned salmon caused symptoms of botulism in 7 of 11 persons who ate small quantities of it. Two of these had only a dryness of the throat with some accompanying difficulty in swallowing; one had, in addition, violent colic and diarrhea. The remaining four exhibited varying degrees of "the classical signs of botulism": asthenia, dryness of the mouth, dysphagia, constipation and the ocular syndrome of paralysis of accommodation, partial amblyopia, choked disk and retraction of the visual fields. Two of the last four cases showed mydriasis, and one a slight unilateral ptosis. All recovered under gastro-intestinal symptomatic treatment and strychnin.

Extended bacteriologic experiments were carried out, and the particular strain of bacillus which was isolated appeared to have low toxin-producing power when compared with that of other strains described in the literature. This was thought to account for the 100 per cent. recovery rate.

HUDDLESON, New York.

BLOOD CREATININ FINDINGS IN FIVE CASES OF CORPUS STRIATUM DISORDER. THEOPHILE RAPHAEL and FREDERICK C. POTTER, *J. Nerv. & Ment. Dis.* **55**:492 (June) 1922.

The prominence of disturbance of muscle tonus and general motility in disorders of the corpus striatum suggested to the authors the possibility of concomitant alteration in muscle metabolism. The cases studied were two of paralysis agitans, two of Huntington's chorea, and one of congenital double athetosis. Blood urea and urine examinations were made, and the patients were kept in bed on a meat-free diet; on the sixth day blood creatinin was determined. The creatinin content in all instances was definitely below normal.

VONDERAHE, Philadelphia.

Society Transactions

THE NEW YORK NEUROLOGICAL SOCIETY

Regular Meetings, Jan. 2 and Feb. 6, 1923.

FOSTER KENNEDY, M.D., *President*

A CASE OF EXTRATHECAL ABSCESS OF THE SPINAL CORD. DRS. ALFRED S. TAYLOR AND FOSTER KENNEDY.

History.—The case here reported was that of a girl aged 15, whose previous history was negative. On November 5, the patient felt a sharp pain over the left lower ribs. Next day the pain continued steadily and she felt chilly. In two more days the pain subsided, but the left lower ribs were sensitive to pressure and a week later a soft swelling appeared at this site. November 17, the legs felt dull and numb and next morning she could not control them and had difficulty in voiding urine. By November 20, there was complete loss of power and sensation in both legs, with loss of sphincter control and slight rigidity of the neck. By lumbar puncture at this time, through the fourth lumbar space, no spinal fluid was obtained, but pure white pus escaped from the needle as it was withdrawn. Her temperature now varied from 101.6 in the morning to 104 in the evening, and she entered the hospital when Dr. Kennedy reported: Motion of the arms and trunk was normal with no stiffness of the neck. The legs seemed to be paralyzed but there was some motion in the flexors of the hip on the right. There was no motion in the toes. On the left the sartorius and vastus internus could be contracted and the toes could be flexed. There was slight rotation of the left ankle. There was complete sphincter palsy. The left abdominal reflexes were absent; the knee jerks and ankle jerks were absent; the plantar reflexes were absent on both sides. There was marked convexity of the outline of the left side over the lower ribs, with tenderness over the five lower ribs. A small tender fluctuating area could be felt over the tenth rib in the anterior axillary line. The left kidney was palpable and tender and there was a large area of dullness to percussion over the left lower ribs, merging with splenic dullness. The lumbar spines were tender to pressure but the spine, as a whole, was not rigid. There was no muscle spasm, so that a primary bony focus in the spine was not present. Sensation was diminished in the first to the fifth sacral areas and this was more notable in the third, fourth and fifth than in the first and second. The lumbar roots were not involved.

Examination of the chest by Dr. Williams showed that the lung was normal except for some compression at the left base. A diagnosis of extrathecal abscess compressing the lower sacral roots was made, the abscess being either metastatic or, more probably, continuous with the subcostal abscess.

Operation.—Nov. 21, 1922, Dr. Taylor removed the laminae of the second, third and fourth lumbar vertebrae. Pus was found between the spines of the second and third lumbar vertebrae on the left side as the muscles were separated from the bone. The epidural fat was infiltrated by foci of pus and a white, thick pus escaped when the dura was pushed away from the left side of the spinal canal. The wound was left wide open and freely drained. An

incision was then made over the left tenth rib where the fluctuating swelling was present. A large amount of pus was evacuated when the skin and muscles had been divided. Pus sinuses led between the tenth and eleventh ribs, and on the inner side of the ribs a large, irregular cavity was palpated running downward to the twelfth rib and upward to the eleventh rib. The lower, posterior part of the cavity seemed to lead toward the lumbar spine and probably this was the position from which direct extension to the spinal canal occurred.

Course.—From the beginning it was necessary to use a catheter. Cultures of urine on Dec. 4 showed *Bacillus coli communis* and later the urine became turbid. The temperature varied from 99 in the morning to 101.6 in the evening for the first twelve days, and showed slight elevations at times till the thirty-fifth day. Cultures from the pus in the spinal canal and chest revealed *Staphylococcus aureus*, from which a vaccine was prepared. Between November 21 and December 26, blood examination disclosed: hemoglobin, 60 to 75 per cent.; white blood cells, 18,000 (polymorphonuclears, 86 per cent.) to 9,000 (polymorphonuclears, 78 per cent.); red blood cells, 3,500,000.

The wound drained well and filled in rapidly with granulation tissue and finally healed. On the second day the sensory disturbance was much less marked and on this day also the patient began to make some movement in the left thigh and increased motion in the right leg. Sensation improved more rapidly than motor power. On the twenty-seventh day she began to pass urine voluntarily in small amounts at irregular intervals.

Jan. 11, 1923, Dr. Kennedy's examination showed: In the right lower extremity: Flexion at the hip was possible but weak; extension was strong; abduction and adduction of the thigh were both good; neither flexion nor extension was possible at the ankle; no movement of the toes could be performed. In the left leg: extension and flexion of hip present, but weak; flexor and extensor movement was present at the knee; at the ankle, extension and dorsiflexion were present; the toes moved freely; eversion was present, but weak; inversion was present and fairly strong. The right knee jerk was brisk, the left knee jerk obtained; neither ankle jerk was present; the plantar reflexes were absent; abdominal reflexes present; pain and touch sensations were normal in the first sacral areas. Over the third, fourth and fifth sacral root areas there remained only a relative loss of pain and touch sensation. There was no loss of sensation of position in either extremity and deep pain sensation in the calf muscles was increased. She had no knowledge of imminent micturition; she had, however, sensation during the act, but lacked control. The rectal sphincter was paralyzed.

A CASE OF VERTEBRAL DISLOCATION: REDUCTION. DRs. ALFRED S. TAYLOR AND FOSTER KENNEDY.

History.—The patient, a woman aged 35 years, on Oct. 25, 1922, was thrown from an automobile, landing on her head. She suffered from a small scalp wound on the back of the head, and loss of power and sensation in the trunk and lower extremities. She also suffered great pain, and frequent muscle spasms of the body, legs and neck. Examination by Drs. Pool and Kennedy showed evidences of a transverse lesion at the level of the sixth cervical segment. There was tenderness over the sixth cervical vertebra. Motion was absent in the legs and trunk. In the arms, the biceps and deltoid, both right and left, were intact. The triceps was partially paralyzed on both sides. The hand and forearm muscles were completely paralyzed on both sides. All deep reflexes were abolished. The plantar reflex was absent on the left side and gave slow flexion on the right (not

a normal, but a pathologic sign). Pain (superficial) and touch were abolished to the level of the sixth cervical segment. Loss of appreciation of temperature was at a level about 3 cm. higher than touch or pain. There was a vague sensation of deep pain in the legs and of movements of the toe joints. Roentgenograms taken with a portable machine, showed extreme dislocation forward of the fifth on the sixth cervical vertebra with some crushing of the articular processes on one side. The neck, in the region between the fourth and seventh cervical was extremely tender, both in front and back, but most tender over the fifth and sixth cervical, where there was some deformity, not exactly determinable because of extreme tenderness.

There was a fullness in the front of the neck at the level of sixth cervical vertebra. Breathing was entirely diaphragmatic. Any movement of the head, neck or body caused extreme pain. The temperature on admission was 97, pulse 64, and respiration 20. General health had always been good and she had always been a hard worker. There was a vague story of trouble with the kidneys many years before.

Operation.—Oct. 27, 1922, an attempt was made to reduce the dislocation without anesthesia. Stretching of the neck preliminary to the reduction was so painful that a moderate amount of ether was used. After stretching the neck for about five minutes the reduction was accomplished without difficulty. Pictures taken on the spot and developed at once, showed that the alinement, both laterally and anteroposteriorly was practically perfect. The articular processes did not seat entirely home, but were in perfectly good position. She was then put in a plaster jacket involving the whole thorax, shoulders, neck, chin and occiput. From the moment of reduction the pains and muscle spasms disappeared and she could move her head laterally and rotate it slightly in her cast without discomfort. Four days after the reduction another picture was taken in order to make sure of the position. There was perfect alinement, even the articular processes having seated home into their normal positions. She made a slow, but uninterrupted recovery.

Course.—The day after admission the temperature reached 99. The day after the reduction of the dislocation (October 27) her temperature reached 101.2 and the next day 101.4; the pulse remained at 82; and respirations, 24 to 28 to the minute. Thereafter her temperature steadily descended and became normal after the eleventh day; her pulse rate averaged from 64 to 78 and her respirations 18 to 24 to the minute. Her urine throughout the period was normal. Her appetite was good and her bowels were kept regular by means of enemas and occasional medication. On the morning after the accident there was slight return of superficial sensation in the legs and thighs that was not present immediately after the accident. The day following the reduction the patient had decidedly more strength in the muscles of the upper extremities, which she had been able to use before reduction had occurred. On the sixth day, following reduction, there occurred definite movement in the toes of the right foot and very slight movement in the great toe of the left foot, although this last was open to question. There was also a faint degree of movement in the thumb of the left hand. On the twenty-second day the patient flexed the third and fourth digits of the right hand. On the twenty-eighth day she moved the second and third digits of the left hand. Movement of the fingers of the right hand is now very much more definite and strong than on the left. There is a slight suspicion of movement in the second and third toes of the left foot. While, at first, breathing was entirely diaphragmatic, within three weeks the intercostals seemed to be working again, although one could not be sure because of the plaster jacket. On the

thirtieth day the patient felt sure that her breathing was almost normal, and one could make out definite excursions of the ribs during respiration. From this time on movement increased steadily. She began to develop the power to raise the left leg off the bed. On the thirty-fifth day, there was very definite increase in strength in the thumb and the first and second fingers of the right hand. On the thirty-eighth day she could move the left big toe very definitely and raise the knee slightly from the bed. On the forty-fifth day another roentgenogram was taken and the spine was found to be still in perfect position. On the forty-ninth day she complained of cramps in the bladder and passed urine spontaneously. On the fiftieth day she voided a large amount of urine voluntarily. From this time on she was able to void more voluntarily (but the passage of urine was felt only on the left side of the urethra). There was also steady, definite, but slow improvement in all muscles in both the upper and lower extremities. On the fifty-fifth day the cast was split and removed long enough to take an impression for the making of a leather collar and support. Afterward the plaster cast was replaced and held with adhesive plaster.

DISCUSSION

DR. FOSTER KENNEDY: In regard to the first case, I would like to have Dr. Taylor's opinion regarding the methods of freeing the extrathecal area from pus and granulation tissue. The pressure is slight, but it seems excessively difficult to drain this area and to get sufficient space for adequate drainage.

DR. ALFRED TAYLOR: The drainage seems to have been satisfactory, and the chances are that the infection has been pretty well overcome. With the inflammatory condition there present, we are bound to have granulation tissue and scar tissue, and we may get pressure later. I believe we should wait several months to see if, in spite of treatment with massage, heat and light, there is permanent cicatrization in the deep tissues, sufficient to cause permanent nerve disturbances. If after that, there is pressure, it would be fair to go into a clean field and try to relieve pressure by secondary operation. As the condition is extradural we have fair hope of success.

DR. FOSTER KENNEDY: In regard to the second case, a word should be said concerning the method of reduction. Dr. Taylor had a set of ropes from the extension apparatus and braced this round his own hips, so that, with his hand under the girl's neck, and leaning his whole weight backwards, he was able to exert a steady pull until he obtained a perfect reduction of the fracture. The entire procedure took about one hour and ten minutes, a period so long that no steady manual traction could have been maintained.

DR. CHARLES A. ELSBERG: I compliment Dr. Taylor on the way in which he has managed both these cases. My personal experience has been less fortunate with extradural cases. By the time the patient is operated on, he is paraplegic, and after operation, remains so. The cases I have seen have usually been due to acute osteomyelitis of the vertebrae. In the second case the roentgen-ray shows an ideal reduction. There is one point, however, to be noted in these cases of dislocation between the fifth, sixth and seventh cervical vertebrae. One sometimes sees an enormous amount of dislocation in patients who improve and get well. I have seen two such cases—one in a high diver, and one in a football player. Both showed a much more marked dislocation than that indicated by the roentgen-ray in Dr. Taylor's patient. This of course, has no reference to the reduction of the vertebrae obtained in this case by Dr. Taylor which promises ideal results.

DR. I. ABRAHAMSON: Was lumbar puncture done in the second case?

DR. TAYLOR: No.

DR. TAYLOR (Closing): I have seen dislocation with marked deformity persisting in a man who made a perfect neurologic recovery after the accident. Later he was brought to me because of a difficulty in locomotion, due to spasticity in the legs. This occurred after eighteen months. It has been my experience that if angulation persists, in time the upper edge of the lower vertebra causes pressure on the anterior columns and spasticity develops as in the case mentioned.

The technic Dr. Kennedy described, I have used in three different cases. It is original. It suggested itself to me in this case. I did the same thing a number of years ago in a boy who made an almost perfect recovery, except for some slight spasticity in the legs in the morning. It is a simple procedure and well controlled; it gives a very satisfactory anatomic result.

DR. FOSTER KENNEDY (Closing): In regard to the first case, it is difficult to decide whether the extrathecal abscess was a direct extension from the front of the chest, or whether it was a metastatic process. There was evidence of compression of the lower chest, bronchial breathing and dulness to percussion; but apart from that, it was fairly clear that had it been a metastatic, lymphogenous, perineural infection from the lower ribs on the left side, an abscess would have occurred in the mid dorsal region, on account of the obliquity of the intercostal nerves.

SENSORY DISTURBANCES IN TUMORS OF THE SPINAL CORD AND MEMBRANES, AND THEIR DIAGNOSTIC SIGNIFICANCE. DR. CHARLES A. ELSBERG.

The report is based on eighty-seven pathologically verified spinal cord tumors of which fifty-nine were extramedullary, fifteen extradural, and thirteen intramedullary.

Dissociation of cutaneous sensibilities is very frequent in extramedullary tumors, almost as frequent as in intramedullary growths; and in the latter all types of sensation are, frequently, equally involved. The more typical disturbances of sensation in extramedullary, as compared with intramedullary disease are the following: Unless there is a loss of all sensation below the segmental level in many extramedullary growths, the maximum of sensory disturbance is in the most peripheral dermatomes and the minimum of disturbances near the level of the lesion. In intramedullary growths, on the other hand, the maximum of sensory disturbance is often at, and below the segmental level. This difference between the sensory disturbances observed in many intramedullary and extramedullary growths can only be explained on the basis that there is a grouping of fibers in the spinothalamic tracts according to limbs and parts of limbs. Clinical evidence supports the view that in the spinothalamic tracts, the most eccentric and most posterior fibers are those that are distributed to the sacral and lower lumbar dermatomes; that in front and somewhat internal lie the upper lumbar and lower thoracic fibers; still further forward and internal, the upper thoracic, and so on.

The types of sensory disturbances described are more characteristic of extramedullary and intramedullary tumors than the dissociation of sensibility that is ordinarily described. The occasional absence of cutaneous sensory diminution over several dermatomes below the segment level can also be explained on the basis of pressure from behind, which has not yet been transmitted to the most anterior portions of the spinothalamic tracts. Similarly, in tumors on the

anterior aspect of the cord there may be no sensory disturbances in the lower sacral dermatomes and the sensory disturbances therefore are very similar to those seen in intramedullary tumors.

The relative degree of involvement of tactile, pain and temperature sensibility varies considerably. Tactile sensation is rarely more affected than pain and temperature sensibility. Pain and temperature sensibility are sometimes equally diminished, at other times temperature sensibility is more disturbed than pain.

There is often marked dissociation between the ability to recognize hot and cold, and in many cases the patient is unable to recognize hot at a time when he is still able to recognize cold; it is also very interesting that cold may be felt as pain over areas in which pain, as tested with a sharp needle, has been entirely abolished.

There is also frequently a dissociation between vibratory and articular sensibility. Vibratory sensation may be undisturbed while articular sensibility is profoundly affected or vice versa; or vibratory sensibility may be profoundly affected on both sides of the body, while articular sensibility is disturbed on only one side.

There are also many variations in the Brown-Séquard picture and in many tumors this type of motor and sensory disturbance is poorly marked. There is generally more motor disturbance on the same side and more sensory change on the opposite side.

In fifteen of the author's series, cutaneous sensation was equally diminished on the two sides of the body, while motor power was more affected on the same side as the tumor, and in five patients both motor power and sensation were most affected on the same side of the body. The author then described the clinical picture which he has called "reverse Brown-Séquard syndrome." He finally stated that in these patients, when bilateral signs become marked, the condition may be one of "bilateral Brown-Séquard" picture.

DISCUSSION

DR. I. ABRAHAMSON: Dr. Elsberg has approached the subject from a standpoint that is neither stable nor valuable. The stage of the disease when the sensory examinations were made was not mentioned. We all know that late manifestations of tumors of the cord, as well as tumors of the brain, are misleading and that conclusions drawn from them are very hazardous. The better method of approach is the anatomic physiologic method, starting with the first signs of the disease and studying the morbid processes in the order of their development. Variations in sensory changes are not accidental or incidental, but are conditioned by the consistence of the tumor and its position in reference to the level and to the circumference of the cord. The softer tumors, with more widespread root signs, cause tract losses of sensation, earliest and severest just below the level of the disease and diminishing in intensity as they spread downward, often sparing the sacral segments; the hard tumor, with limited root signs, causes the maximum tract sensory disturbances over the lowest sacral distribution with diminishing intensity as we ascend toward the level of the disease.

The position of the tumor with reference to the cord is equally important: lateral pressure causes early and more marked Brown-Séquard syndromes; anterior or posterior pressure causes disturbances of the posterior column sensibility early and intensely and, only later in the disease, irregular disturbances of the pain and temperature sensibility resembling the Brown-Séquard picture.

There are other dissociations between sensory and motor disturbances, and these also depend on the site and degree of the cord compression.

I believe that the signs and symptoms are due, primarily, to circulatory changes within the cord, caused by pressure and interference with the various circulations, and not by the direct pressure on contiguous portions of the cord. I have postulated lamellation of the spinothalamic and other pathways for years; but the type described by Dr. Elsberg, bearing a relationship to the circumference of the cord and not to vascular supply, did not coincide with this view of the matter. The paper of Dr. Elsberg, while interesting, is apt to create confusion in the mind of all but the most expert. The confused sensory charts require proper keys or small diagrams to illustrate the position, age and density of the tumor.

DR. G. ROSETT: How frequently did Dr. Elsberg find sensory and motor disturbance on the same side? In going over the records of a number of cases with Dr. Tilney, I noted a number of cases of lesions of the upper part of the spinal cord in some of which the sensory and motor disturbances were on the same side.

DR. FRIEDMAN: I was glad to hear Dr. Elsberg dispose of the notion that the so-called syringomyelic type of sensory disturbance is found only in intramedullary tumors. We have been making observations similar to those of Dr. Elsberg, with regard to the question of lamellation in the spinothalamic pathways, in the neurologic service of Dr. Kennedy at Bellevue Hospital. We have also found that in extramedullary compression of the cord from side to side, there is the type of sensory change spoken of by Dr. Elsberg. We have termed this a "long tract pressure sign." That the fibers from the lower dermatomes range themselves peripherally in the cord, is also shown by the type of sensory change seen in those tumors of the cord which exert pressure anteroposteriorly. In these cases we get exclusion of the sacral areas with the type of sensory change seen in intramedullary tumors.

I might also suggest that in the few cases in which the motor and sensory signs seem to be on the same side, the mechanism might be that of contrecoup, the motor signs being due to homolateral injury of the pyramidal tract and the sensory changes the result of lesion of the opposite spinothalamic pathway through jamming of the cord against the spinal column.

DR. ELSBERG: When there is a tumor of the anterior aspect of the cord near the median line, one is in doubt whether it is an intramedullary tumor. When the tumors lie mesially on the anterior aspect of the cord, the disturbance is rather of the intramedullary than the extramedullary type.

SOME REFLECTIONS ON THE MODERN SCHOOL OF DUTCH NEUROPSYCHIATRY. DR. SMITH ELY JELLIFFE.

The modern Dutch school of neuropsychiatry probably embraces the greatest number of eminent workers in this field at the present time. The work of the late Dr. J. K. A. Wertheim Salomonson, Rector of the University of Amsterdam, embraced a very wide field, covering a knowledge of medicine, physics, neurology and roentgenology; in the latter branch he was esteemed the chief leader in Holland. He had a lifelong interest in neurology, and devoted considerable time to this subject in later years. His chief researches dealt with the electric degeneration of the peripheral nervous system. Much of his research work was directed toward clinical application, and of this may be mentioned studies on clonus and tetanus, psychogalvanic reflexes, shortening reflexes and measure-

ment of chronaxia. He was also interested in the string galvanometer and applied it to neurological findings.

Prof. C. Winkler, the present Dean of Dutch neuropsychiatry, and director of the University Clinic at Utrecht, is head of a modern psychopathic hospital, and author of a number of works which form a mine of neurologic learning. Winkler early devoted himself to neurology and psychiatry and for many years strove to educate the university in the need to provide special chairs in these subjects. For a long time lack of funds hampered this project and political machinations prevented its fruition, but his idea has now been realized. His life was devoted to the advancement of neuropsychiatry, and he became particularly interested in brain function, at one time making a success as a brain surgeon. During his career Winkler taught many students. He founded the neuropsychiatric society in Amsterdam and became a member of the Royal Academy of Sciences. He founded a Central Institute for Brain Research, of which Ariëns Kappers was made director. At this time also a Central Brain Commission was founded. His work embraced surgical experience, together with anatomic and experimental knowledge, but these were always subordinated to the ideal of therapeutic results. His thought was deep and far-reaching, and his research patient; his therapeutic results were striking. During the early days of the war the University of Utrecht, after many delays, founded a professorship of neuropsychiatry, a goal which Winkler had striven to accomplish for thirty years and Winkler assumed charge of this department. In his works Winkler shows remarkable catholicity of thought, and originality of conception. He is bound by no "school" and is preoccupied by the exploration of that difficult subject, the relation of the "soul" to the body. He is an advocate of the theory that the human body is a capturer, transformer and deliverer of energy.

In considering the enormous field of Winkler's research work, time only permits of the mention of one, perhaps the most interesting. This is his work on the eighth nerve, which he regards as originally a cutaneous nerve and belonging to the oldest group of proprioceptive systems, reaching back in time to the marine habitat. Its functions were first adapted to water shock and later to other forms of pressure. The basis for these studies was work on marine molluscs in which the statocyst organ, influencing the equilibrium of the animal, is the point of departure for a functional study of the labyrinth. Winkler concludes that the labyrinth is the most ancient of sensory organs. He traces the development of vestibular and cochlear nerve components, and the gradual acquisition of sound wave components to the stimuli of aquatic nature. Thus the hearing mechanism has evolved out of the primitive water shock mechanism of the statocyst to respond more and more to air shock reflex activities. A further development of cochlear responses is along the line of audition, rhythmic movement reflexes (dancing, singing) and finally speech activities. Thus hearing and speech conserve, as a heritage of ancient function, the power of reproducing by reflex pathways the phenomena which have given place to the stimulus.

Another important phase of the work by the Dutch school, is that on muscle tonus, in connection with which must be mentioned the names of deBoer, Boeke, Dusser, deBarenne, van Rijnberk, Winkler, Jelgersma, Magnus and Kleijn, and Brouwer.

There has been considerable research, and also difference of opinion among Dutch workers, regarding the so-called double innervation of striped muscle tissue. Boeke established the existence of small neurofibrillar end rings, and end nets, at the end of fine non-medullated nerve fibers, differing entirely from

ordinary end plates. While the morphology is fairly well established, the physiology of these plates is still under active discussion. Plastic mechanical factors, as opposed to the view of chemical nutritive factors, are the main contentions. Pekelharing has mainly studied the latter viewpoint, while deBoer has been one of the most active in claiming the existence of plastic tonus function. DeBoer, experimenting along the lines of pharmacodynamics concludes that skeletal muscle tonus has a definite peripheral autonomic reflex arc component. Numerous correlates would be the outgrowth of such a conception, embracing many experimental and clinical possibilities.

Passing on to the work of von Rijnerk on muscle tonus, the theory of this worker was that there was a vegetative supply to striped muscle, and this was utilized to account for Vulpian's lingual paradox. This theory may help to explain the muscular tonic phenomena following facial palsies, and explain the "foot phenomena" of Sherrington.

Certain phases of the same problem have been extensively studied by Magnus and Kleijn, of the department of pharmacology of the University of Utrecht. They have concerned themselves with the study of decerebrate rigidity and find that the cerebellum plays no part in this syndrome. These workers have separated the labyrinthine from the neck reflexes; the former arise from stimulation of the otoliths, the latter from proprioceptive nerve endings of the cervical muscles. Kleijn describes four components in the postural reflex: (1) head labyrinthine reflex; (2) body wall reflexes influencing head positions; (3) neck muscle reflexes; and (4) trunk and limb muscle reflexes.

Prominent among the workers on cerebellar physiology and morphology are Elliot Smith and Bolk, both of whom are innovators and originators of a new concept. The comparative anatomy of lower species is studied, working from below upward in the scale of organisms, and the conclusion is reached that the several parts recognizable in the mammalian cerebellum have definite functional significance and represent areas of central control over definite motor performances in the body. Bolk has mapped out the various divisions and subdivisions of the cerebellum and finds striking variations between different organs. He believes there is functional variation related to structural variation, since the form is dependent on function. He traces the relationship of various parts of the cerebellar cortex to the body movements (bilateral synergic, or unilateral synergic) with which they are associated. The work of Bolk, preeminently anatomic, has been confirmed by other workers, through animal experimentation.

Jelgersma, professor of psychiatry in the University of Leyden, studied paralysis agitans and has demonstrated alterations of the striated fiber system. His studies on the cerebellum have shown that only two feeling qualities are localizable in this organ, namely: deep sensibility of muscle and joint; and secondly, tonus and equilibrium. Time will show whether these views are correct. Jelgersma has written a masterly text-book on psychiatry. Like many of the younger Dutch neuropsychiatrists, he is greatly interested in psychoanalysis.

Ariëns Kappers is director of the Central Institute for Brain Research, and a pupil of Winkler. He has done considerable work on the comparative anatomy of the nervous system. His thought is along the line of conception of organizing synthetic function of the nervous system in response to cosmic stimuli, and he holds that a structural evolution is going on within the nervous system, which he terms neurobiotaxis. He believes function is primary; structure is secondary and variable according to the needs of the organism.

Dr. Jelliffe then took up the work of several other members of the Dutch school and gave illustrations from their works, particularly B. Brouwer, H.

and G. C. Bolten, and L. and H. Boumann. Among other things Dr. Jelliffe pointed out the contributions of Brouwer to the study of the cerebellum and the olives, and drew particular attention to his work on the oculomotor nuclei. Brouwer's utilization of the evolutionary history of neurologic structures makes his work particularly valuable. His study of multiple sclerosis from a phyletic viewpoint was mentioned and illustrated.

Dr. Jelliffe then called attention to the work on the sympathetic nervous system by the Boltens which lay close to his (Dr. Jelliffe's) interests, and then gave a very short sketch of the psychiatric opportunities in Amsterdam with the two psychiatric clinics.

DISCUSSION

Dr. WALTER M. KRAUS: Dr. Jelliffe, in his delightful review of the work of the Dutch school, has focussed our attention on subjects with which we ordinarily would not have come in contact. He has reviewed a number of interesting neurologic topics of great value. I suppose that research activities in neurology could be divided into three parts: In one, the subject is approached from the viewpoint of formal psychology and, with practically no organic basis, an attempt is made to unravel the most complicated reactions of the human brain. Another group devotes itself to the study of facts as they occur, without being much concerned with their significance in terms of past or future evolution. The third group is interested in tracing the evolution of the human nervous system. As Dr. Jelliffe has emphasized, this last method is essential to the solution of most problems in neurology.

While in Amsterdam, I had a short interview with Dr. Kappers, which was very stimulating. His collection of comparative anatomic material is the finest in the world, and the opportunities for research, which this vast collection offers, would keep a score of men busy for a long time. For those who find it impossible to go and work with Dr. Kappers, his textbook on comparative neurology and his papers on this subject will constitute a source of much information and inspiration to work.

To mention a few things which may be found in his book to stimulate interest in the phylogenetic significance of certain parts of the nervous system: The pyramidal tract, which we have dignified with so many functions, is present only in mammals. Bearing this in mind and also recalling that the pyramidal tract forces its way through the striatum, we derive some notion of a reason for the frequency of hemorrhage from the blood supply of the pyramidal system in the capsular region. Obviously, these blood vessels must be comparatively recent from a phylogenetic point of view and may be expected to be more easily irritated by toxins and thus more easily diseased. In another place Kappers states that in the lower mammals the pyramidal tract appears in the posterior column. Other fibers in these latter columns convey sensation through several relays to the cortex. One wonders, in the light of Kappers' theory of neurobiotaxis, why the pyramidal tract wandered from this position to its present position in the lateral columns.

The nucleus of the twelfth nerve in lower animals without a tongue lies on the ventral side of the medulla. After the tongue appeared, the nucleus assumed a dorsal position in contiguity with the sensory nuclei concerned with the various sensations received from the tongue. This is a beautiful example of neurobiotactic activity. In order to understand the functions of the nervous system as they appear in man it is essential to study their phylogenetic history.

PHILADELPHIA NEUROLOGICAL SOCIETY

*Regular Meeting, Feb. 23, 1923*DR. C. M. BYRNES, *President*

A CASE WITH UNUSUAL MENTAL SYMPTOMS. DR. E. V. EYMAN.

History.—A married man, aged 29, was admitted April 17, 1922, to the Pennsylvania Hospital for Mental and Nervous Diseases. His family history was negative. He had a small depression in the left parietal region near the vertex caused by a stone at 8 years of age. He had acute rheumatic fever in October, 1917, while in the navy and has never felt well since. He suffered a fracture of the left arm and a laceration of the scalp over the old depression in November, 1917. Severe headache started after he received the injuries recited and persisted constantly till Feb. 9, 1923. One month after the onset his memory became poor, but he performed his duties on the ship till discharged in February, 1919. At that time he was irritable, restless, had diplopia occasionally, and fell asleep readily. He attempted to resume his duties as an electrician, but his poor memory and absent-mindedness caused him to receive several severe shocks. He then became a masseur. The headache continued and his memory grew worse. He had difficulty in finding his way home, sometimes wandering for hours, but eventually reaching his destination. He became sullen, suspicious, showed increased sexual excitability, developed ideas of marital infidelity and said he had "imagination."

Physical Examination.—The general examination was negative except for the usual signs of aortic regurgitation. When tested for Romberg's sign he always started to fall and then staggered backward and to the right; when walking blind-folded, he turned to the right in a large arc. He responded to commands very slowly. Spontaneous movements were often quickly performed if associated with every-day acts. The face was expressionless and mask-like. The cranial nerves, including the olfactory nerves, were normal with the exception that both retinas were moderately congested, with slight blurring of the nasal disk margins. The neurologic examination otherwise was negative.

Laboratory Findings.—Examination of the urine showed albumen, hyaline and granular casts. The Wassermann test and spinal fluid were negative. The roentgenogram of the skull was also negative.

Mental Examination.—Memory for recent and remote events was almost nil. He was not depressed, but extreme retardation in cerebration was present; it took him ten minutes to say that a nickel was money, fifteen minutes more to say it was 5 cents, and five minutes to say it was used to buy things. It took him forty-five minutes to find his way back to his ward. He did not recognize his wife or child, but in spite of all this he could play a good game of checkers, pool or baseball, and find his way to the toilet. He occasionally experienced difficulty in finding his room. His mental condition resembled none of the psychoses.

Course.—During his hospital residence the patient's memory grew gradually worse, as did his retardation, until February 9, when he suddenly cleared up. For a time after his sudden recovery, his memory for the past six years consisted merely of a few hazy patches, but he has gradually improved in this respect until his memory seems good for everything except the period of his

hospitalization from April, 1922, to February, 1923. His memory for dates for the years immediately following his discharge is still somewhat uncertain. He was seen by many physicians during his illness and a list of some of the diagnoses then made follows: (1) traumatic psychosis; (2) frontal lobe tumor; (3) post-encephalitic condition; (4) manic-depressive depression; and (5) hysteria.

In favor of frontal lobe tumor were the constant headaches, great memory defect, difficulty in finding his way home, and the gradual progression of the condition. Against it were the integrity of olfactory sense and absence of positive changes in the fundus. Tumor is of course ruled out by the patient's recovery. In favor of a post-encephalitic condition was the history of diplopia, drowsiness, the mask-like face, and the slowness of movement in response to command. The suddenness of the recovery seems incompatible with this diagnosis. In favor of the diagnosis of traumatic psychosis we have a fairly definite history of onset following severe injury, but the slowness with which the patient's condition reached its peak does not seem compatible with this diagnosis. Favoring hysteria are the absence of definite neurologic findings, some inconsistencies in the mental symptoms, and the sudden recovery; but again the gradual onset, and the very prolonged duration of a monotonously colorless picture seem unusual. In favor of manic-depressive depression, is the presence of retardation; but the retardation did not include habitual acts such as eating, and there was no very marked depression. My own diagnosis previous to the recovery was a post-encephalitic condition; now it is hysteria.

DISCUSSION

DR. ALFRED J. OSTHEIMER: This man is a naval ex-service man and I have had him under observation for over a year. I shall add a few points to the history given by Dr. Eyman. The man has a marked depression of the skull, but the navy records show that he had this depression when he first enlisted thirteen years ago. I conclude, therefore, that the old fracture of the skull has had nothing to do with his present condition. There is no doubt that he had a severe accident during recent service, but I think it was not an etiologic factor in his recent mental condition. He had an attack that was called influenza in 1918, and, early in 1919, developed these mental symptoms, with irritability and complete change of character. This man had a very good record in the navy. After he came home he was unable to retain his position as electrician because of irritability, dulness, lethargy and lack of ability to concentrate on his work. Dr. Eyman says that one of the facts that is not compatible with a diagnosis of a post-encephalitic psychosis is that his mental condition has cleared up so quickly. He did not clear up over night. The whole description, I think, corresponds to the rather bizarre character of a post-encephalitic psychosis, notwithstanding the fact that he has recovered rather promptly. I am inclined to believe that the man has had a post-encephalitic psychosis, a long time after the acute attack to be sure, but comparable organically to the parkinsonian syndrome seen so frequently now and manifesting itself one, two or even three years after the acute infectious disease, probably consequent on degenerative structure changes. It may be that I am somewhat influenced by the present policy in the U. S. Veterans' Bureau of giving the man the benefit of the doubt in order to be sure to give him justice, and therefore compensation for a service-connected disability. In this case it seems to me that there is a question of doubt, and when there is a question of doubt, it should be resolved in favor of the man.

HEMICHOREA OF SUDDEN ONSET IN WHICH THERE WERE NO OTHER SYMPTOMS. S. F. GILPIN, M.D.

The following case is presented because, in the six months that the patient was under observation, there were present only choreo-athetoid movements in the left arm and leg, although in the beginning the movements were also present in the left side of the face. Repeated examinations demonstrated no other symptoms. Such a case is most unusual and difficult to localize. It illustrates, however, that limited lesions may produce athetoid movements only.

History.—A white woman, American by birth, 57 years of age, a widow with one son who is in good health, has a negative family history. She was in the Jefferson Hospital twelve years ago for some inflammatory condition of both knees and was operated on in the University Hospital six years ago, an ovarian cyst and the right ovary being removed. For several months she has been troubled with frequent micturition and swelling of the ankles. The present illness began suddenly Aug. 31, 1922. While going from one room to another she felt dizzy and swayed against the stair-rail. Immediately afterward she walked two blocks to my office, and then presented the choreiform movements as seen at present.

Physical Examination.—The patient shows no motor weakness, sensation is normal and the pupils, reflexes and laboratory examinations are negative.

ERB'S TYPE OF SPINAL SYPHILIS PRESENTING DIFFICULTIES IN DIAGNOSIS. S. F. GILPIN, M.D.

The patient, a white woman, aged 52, was admitted to the hospital from the dispensary Dec. 15, 1922. In spite of a negative Wassermann test she had been considered syphilitic and was given mercury by inunction; but by mistake she used double the amount of mercury ordered. In four days she came to the clinic with such severe salivation that she was admitted to the ward for treatment. Her chief complaint was pain beneath the shoulders, in the groins, the balls of the feet, the heels and pain of shooting character in the backs of the legs. She is married but has never been pregnant and has been an actress. She has always had good health, except for the present trouble which began five years ago and has become much worse during the past six months. For the past four months she has been unable to walk in the dark for fear of falling.

Physical examination reveals ataxy of gait and station. The pupils are equal but react sluggishly to light. The knee-jerks are increased, but there is no clonus nor Babinski sign. Repeated examinations of the blood and spinal fluid have yielded negative Wassermann tests; four cells were reported in the spinal fluid at one examination. A second laboratory reported a doubtful (+) Wasserman reaction in the spinal fluid. Roentgen ray examination of the dorsal and lumbar spine was negative. She rapidly became worse, her legs became so weak that she could not get out of bed and she developed marked distention of the abdomen, although surgical and gynecologic examinations proved negative. The distended abdomen was thought to be due to gas, secondary to relaxed or paralyzed intestinal muscle. Believing the case to be syphilitic in spite of the negative Wassermann reactions, she was subjected to the therapeutic test of rapidly increasing doses of iodids. She tolerated the treatment well and rapidly improved. At present she is taking 100 grains (6.3 gm.) of sodium iodid three times daily; she has no pain, can stand and walk without ataxia and her knee-jerks are normal. Did this patient have syphilis? If so why was the Wassermann test persistently negative except for

a + reaction on one occasion? If she does not have syphilis, why did she improve so marvelously under iodids? Is the old fashioned therapeutic test more reliable in the diagnosis of a difficult case of syphilis than the Wassermann test?

DISCUSSION

DR. CHARLES K. MILLS: Regarding Dr. Gilpin's first case, he reported no loss of sensation on either the hemichoreic or the other side. The case is a difficult one for diagnosis. At first sight, I thought it might be an instance of a lesion of the superior cerebellar peduncle, but in the absence of impairment of sensation, it is not possible to explain the case in this way. The only explanation I can see is that there is a focal lesion in some part of the striate body.

DR. FRANCIS X. DERCUM: My own feeling was that, in this case of hemichorea, there was a limited lesion of the striate body and that the movements were in a sense comparable to those which we occasionally see in encephalitis. Several years ago I described two cases of hemichorea of rather rapid onset.

The second case of Dr. Gilpin is interesting because the woman had a pronounced paraplegia. I regarded the case as one of Erb's form of spinal syphilis. The laboratory reports came in repeatedly negative, but in view of the very clear clinical findings I set these reports aside. My attitude in general, in regard to the Wassermann reports, is to assign to them a secondary importance, especially when the clinical findings are positive. This case is remarkable because of the brilliant results following the administration of the iodids in massive doses. This woman was almost completely paraplegic, she stumbled and fell when she attempted to walk but at the present time she walks with a normal gait. We have fallen in a measure under the spell of arsphenamin administration and frequently neglect the massive administration of the iodids. Here is an instance of a result not infrequent among the older neurologists, a brilliant recovery from the use of large doses of the iodids, especially when it is employed in early cases.

DR. W. G. SPILLER: It is interesting to note that S. A. Kinnier Wilson, in a paper recently published, states that the best treatment for syphilis of the nervous system would be the intradural injection of mercury if we had a form of mercury that was not irritant. Negative Wassermann findings do not always exclude syphilis, and we do not always obtain positive findings in tabes, which is a syphilitic disease.

DR. S. F. GILPIN: As to the treatment of the second case, I hope that the time will come when the medical profession will realize that, in the treatment of neurosyphilis, it is not necessary to inject anything into the subarachnoid space to get results. It is now four years since I showed a case of tabes to this society, in which the Wassermann reaction and cell count became negative. The patient's gait and station became normal, although she had been so ataxic that she required assistance to come to the clinic. The disease had existed four years before she came under treatment. She has not received treatment for five years and the Wassermann reaction and cells remain negative. The patient had mercury, by inunction only, with systematic drainage of the cerebrospinal fluid. The results in her case have been duplicated in others.

ILLUMINATING GAS POISONING: REPORT OF A CASE PRESENTING AN UNUSUAL SEQUELA. GEORGE WILSON, M.D.

History.—F. S., a white man, aged 60, was admitted to the Episcopal Hospital on Dec. 25, 1922, in an unconscious condition. The night before he was

visiting in a strange house and is clear about events until 10:30 that evening, when he became unconscious and remained so until late the following afternoon. According to the history brought by the police, he was found in a gas-filled room. When he regained consciousness, he complained of pain in the right hip and showed evidences of bruises on the left knee and right temple, these being apparently of small moment. Four days after the occurrence recited he awoke from a sound sleep and found that he could not move the right foot, although he could move the knee, and that the entire lower extremity was greatly swollen. At no time has he had any pain except a pain in the right hip, which was first noticed when he regained consciousness. He has had no headache, no diplopia, and no disturbance of the sphincters.

Physical Examination.—Jan. 9, 1923: Pupils, cranial nerves, both upper and the left lower extremities are normal. The abdominal reflexes are absent, probably because of obesity. The entire right lower extremity is much swollen, the swelling being due to edema that pits on pressure. All movements of the right foot are lost, but movements of the right knee and hip are well performed. The right lower extremity lies in a position of helpless eversion, a position frequently assumed in fracture of the hip. The sense of position is severely disturbed in the toes of the right foot. Touch, pain, heat and cold sensibilities are lost in the right extremity as high as the lower third of the leg; from that point to the knee there is diminution of these forms of sensation, which are normal in the thigh. No disturbance of sensation is present in the left lower extremity. Both patellar reflexes are present and active; the Achilles reflex is present on the left and absent on the right. Plantar stimulation produces flexion on the left and no response on the right. Electrical reactions on Feb. 20, 1923, showed feeble reaction to faradism in the affected muscles. Dr. Fewell reported no changes in the optic nerves. Dr. Bromer made the following roentgen-ray report: There is slight roughening of the articular borders of the head of the right femur and of the acetabulum; marked bone proliferation and roughening of the joint surfaces of the tenth, eleventh and twelfth dorsal, and of the first and second lumbar vertebrae are present.

On the day of admission to the hospital, the red cells numbered 6,400,000; white cells, 22,000; and hemoglobin, 95 per cent. Four days later his white cells had dropped to 14,160 and two weeks later were normal. Examination of the urine on two occasions showed the presence of albumen and casts. The blood Wasserman reaction on December 29, was negative.

The localization of the lesion in this case seems to be in the cord in the epiconal region on the right side. This would explain the paralysis and the loss of sensation. The edema was probably due to vasomotor paralysis.

DISCUSSION

DR. WILLIAM G. SPILLER: I do not think Dr. Wilson has sufficiently excluded the possibility of vascular thrombosis in this case. He and I have been interested in phlebitis of the upper limb, and phlebitis of the lower limb is well recognized. In some cases the thrombus forms in an artery. The symptoms probably are not produced by pressure of the thrombotic vessel, but by impairment of the circulation to the peripheral nerves.

DR. GEORGE WILSON: This case does not impress me as one of disease of either the femoral artery or femoral vein. It obviously is not a disease of the artery because of the edema. The single fact that the man did not have pain rules out the possibility of phlebitis.

MOTHER AND DAUGHTER PRESENTING SYMPTOMS OF A DEGENERATIVE STATE OF THE CENTRAL NERVOUS SYSTEM.

ALFRED GORDON, M.D.

The mother aged 56, was in good health until seventeen years ago, when she first observed an awkwardness in the fingers of one hand which was followed by a gradual wasting of the muscles of the hands. The atrophy slowly involved the lower extremities and finally the muscles of the thorax and of the face. When first examined about ten years ago she presented, with one exception, the same condition she now shows. The exception consists in the state of the patellar tendon reflexes which were then increased, but are now abolished.

At present the picture is that of progressive muscular atrophy of the spinal type. The tongue presents a fine tremor; it also is somewhat flabby, but its power is still preserved. The jaw reflex is present and slightly increased. Sensation is normal all over the body and the sphincters are intact. The eye examination shows a convergent right eye; the pupils are 2.5 mm., the right slightly irregular, and both react sluggishly to light and accommodation. The retinal veins are somewhat tortuous, otherwise the eyegrounds are negative. She has a steppage gait. There is slight incoordination in walking, very likely because of the atrophic condition of the extensor muscles. Lately she has developed some difficulty of deglutition and of respiration.

During her entire illness she has not suffered from pain. The blood Wassermann reaction is negative. She has had four children, three of whom died at an early age.

The living child is a girl of 16, who has always had good general health, but noticed difficulty in walking several months ago. Physical examination reveals: a slight Romberg sign; the gait is somewhat difficult, because of the equinus position of the feet which are short, the toes being claw-like because of forced extension. The patellar and Achilles' reflexes are lost. A Babinski sign is present, more frequently on the right than on the left side. In every other respect the patient is normal. The case evidently belongs to the category of Friedreich's ataxia. The characteristic feet, the slight ataxia of station, the absence of tendon reflexes, the presence of the toe-phenomenon, the onset of the disorder at the age of puberty—all speak in favor of the above diagnosis, although there is no nystagmus, involvement of speech, nor ataxia of the upper extremities. It may be of interest to note that the appearance of the disorder dates from the time she commenced to use her lower extremities to excess, being obliged by her occupation to stand almost the entire day. This is in conformity with Edinger's "Ersatz-Theorie"—the theory of exhaustion, which explains the onset of the symptoms of Friedreich's ataxia at the age of puberty.

The occurrence of the two cases in the same family demonstrates a lack of development or of resistance of the central nervous system—in the mother of the ganglion cells and in the daughter of the tracts of white matter.

FOUR CASES OF POST-ENCEPHALITIC SEQUELAE. A. M. ORNSTEEN, M.D.

CASE 1.—A married woman, aged 26, in 1918 had influenza, during which she was drowsy. Two years later, during pregnancy, she was markedly lethargic in spells, falling asleep in the midst of the most interesting environments. This continued for six months, the lethargy being attributed to the kidney toxemia

of pregnancy. On the delivery table, the right leg began to tremble and has not stopped since. From that time there has been a steady progression of the above symptoms.

Between the attack of influenza and the lethargy during pregnancy she was perfectly well. She presents a parkinsonian attitude, facies and gait. The right arm and leg are hemiparetic and hypertonic, with increased deep reflexes and hemitremor of the paralysis agitans type, but without Babinski's sign. The left arm and leg are slightly hypertonic but not paretic. She is obese and has gained 80 pounds, her present weight being 235 pounds. She has polyuria, polydipsia and a large appetite. She is always drowsy, and there is mental apathy. The pupils are large, unequal in size and irregular; the light reaction is greatly impaired, the convergence contraction much better. At one time the pupils were of the Argyll Robertson type; of late the light reaction has improved. There is no defect in the visual fields. Lately she has spells of involuntary crying. The sugar tolerance is definitely lowered. The blood and spinal fluid Wassermann reactions are negative.

CASE 2.—A man aged 30 who, three years after a mild attack of influenza in 1919, in which he was lethargic, began to develop the typical parkinsonian picture without tremor. The long interval of apparent normality is of interest.

CASE 3.—A woman aged 25, also three years after a mild attack of encephalitis, began to develop a typical parkinsonian syndrome with slight right-handed tremor, the three year interval being free of symptoms. Immediately preceding the onset, the patient received a severe mental shock, to which her present condition is attributed by the family; perhaps this shock precipitated the symptoms.

CASE 4.—A girl aged 4, who is unable to stand or walk, the legs being markedly ataxic and atrophic. The reflexes are exaggerated and the plantar responses flexor in type. The upper extremities are also ataxic and are held usually abducted with the hands clasped. The bladder is without voluntary control. The facial expression is idiotic; there is constant gnashing of teeth because of bilateral masseter spasm, and much drooling of saliva. The child is unable to talk. There is a tendency to internal strabismus. The right lower extremity is constantly being thrown about in gross purposeless movements, associated with much snorting and marked irregularity of respiratory rate and rhythm. In the ninth month of the gestation period, the mother had had encephalitis with much drowsiness. The child was born ten days before the expected time with no difficulty and not asphyxiated. She has had no convulsions. The child learned to walk and talk in normal time and at 16 months was able to say such words as "pretty thing" and to walk two blocks by holding on to the coach. At 18 months she began to retrogress, and in three months had reached the present state. It is presumed that the child had encephalitis in utero during the mother's attack and that a period of normality of eighteen months elapsed, such as we see in adults, before the development of the sequelae.

DISCUSSION

DR. ALFRED J. OSTHEIMER: I am much interested in Dr. Ornsteen's cases, on account of the length of time between these recrudescences (these lightings up of the old condition by some new infection or by the strain of pregnancy hitting, as it were, the locus minoris resistentiae) and the original acute attack. It may be that in my work in the U. S. Veteran's Bureau I am particularly interested in this condition because scientifically it brings out the fact that the original infection of ex-service men, which might have been so slight as to

have been overlooked, may have occurred in service and then it becomes possible to connect the sequelae with the service. Therefore, from the standpoint of professional opinion, it is a very great aid in bringing about justice to the ex-service man.

DR. W. G. SPILLER: Clinical recovery does not necessarily mean pathologic recovery. Schaller and Oliver have reported a case of chronic encephalitis extending over fourteen months and followed by necropsy. Some cases of encephalitis assume a chronic type, possibly with remissions. We may not conclude always, because the patient seems clinically well of encephalitis, that all pathologic changes have disappeared. It is possible that some patients carry inflammatory lesions in their brain for one or two years, or even longer, and it is not necessary to believe that the organism persists in the tissues. Cases are reported where symptoms have recurred after a considerable time following what was apparent recovery from encephalitis.

DR. F. X. DERCUM: The term influenza in these cases ought not to be confused with the original disease itself, namely encephalitis. There has been a tendency at times, I think, to confuse the early symptoms of encephalitis with those of influenza and again to correlate the two affections in such a way as to make encephalitis appear as a sequel of influenza. This is something which the facts do not justify. We need only to recall the history of the appearance of encephalitis in Vienna in 1916. It is noteworthy that there had been no influenza in Vienna for a long time previously. I think Dr. Spiller's view is the philosophic and scientific one to take. These patients are not well when they have recovered from their early symptoms. An antitoxin is early developed which results in a relative immunity and partial recovery, but sooner or later they exhaust their defensive resources and the symptoms recur. The same thing is seen in general paralysis. A case is rested and fed and a remission ensues, but sooner or later there is a recurrence of the symptoms.

DR. A. M. ORNSTEEN: The infrequency of these manifestations before the epidemic of influenza in 1918 as compared with the frequency now, cannot but make us consider the probable causal relationship between the original epidemic and the many cases of these syndromes that we see today.

"NAMES AND THINGS IN PSYCHIATRY." CHARLES W. BURR, M.D.

This paper will be published in full elsewhere.

CHICAGO NEUROLOGIC SOCIETY

Regular Meeting, March 15, 1923

H. I. DAVIS, M.D., *President, in the Chair*

A CLINICAL CASE PRESENTING INTERESTING MENTAL PHENOMENA. DR. GEORGE W. HALL.

This report will be published elsewhere.

MYOPATHY AND AMYOTONIA IN THE SAME PATIENT. DR. ALBERT B. YUDELSON.

History.—K. M., a white American girl, aged 10, who was first examined in July, 1920, was born when her father was 34 and her mother 32. The father

had a severe attack of typhoid fever in 1905 but made a good recovery. The parents are both well and there is nothing of significance in the ancestral history. The patient is the eighth child. The first child was born dead, ten days after the mother slipped and fell; the second, seventh and twelfth children are living and well; the third and fourth were born prematurely and lived only three days; the ninth child died with "spasms" at the age of $3\frac{1}{2}$; the tenth pregnancy was with twins, one of which is alive and well, the other died of influenza; the twelfth and last pregnancy resulted in a miscarriage in the third month.

The patient was a bright healthy child and began to walk and talk at sixteen months. She had whooping cough at 2 and measles at 5 years. When $2\frac{1}{2}$ years of age, it was noticed that she did not walk as well as formerly and dragged the left leg. After walking less than a block, or when running across the floor, she would "flop down" to a sitting position; she did not fall, but just sat down suddenly. In getting up from the floor, she "stood on all fours" before balancing herself on her feet. The condition has gradually been getting worse; she has progressively walked less and fallen more often. Her hands and arms twitch and she frequently drops things. She is bright, holds her own with the other children, has ridden to school for the past two years and is now in the third grade. She can sing, read, write and sew.

Examination.—Development is asymmetrical but she is moderately well nourished, of good color, 4 feet $1\frac{1}{2}$ inches (125.75 cm.) tall and weighs 51 pounds (23 kg.). The left side of the thorax appears smaller than the right; there is a scoliosis with convexity to the right and a moderate kyphosis of the middle third of the dorsal spine. The scapulas are winged; the left shoulder droops and the left arm appears correspondingly longer than the right when the patient stands erect.

The musculature in general is flabby and very deficient in tone, especially on the left side, and there is corresponding weakness. With the dynamometer the left hand registers 22, the right 26; the left foot registered 3, the right 5. This indicates marked weakness in the lower extremities. The neck muscles are the firmest and strongest in the body. There is a coarse tremor of the hands, which is increased on intention, and myoclonic movements are observed in the shoulder and gluteal muscles. There is no fibrillary twitching.

The arms can be raised above the head in full extension and adduction, abduction, flexion and extension of the shoulders are free and full. Flexion is good at the elbows, wrists and fingers; the thenar and hypothenar eminences are normal; pronation and supination are normal, though slower on the left than the right. The flexors generally are stronger than the extensors. With her hands the patient performs all purposive movements well except that they are slow, feeble and tremulous. Writing shows waves in the upstrokes.

Hypotonicity is extreme and much more marked in the lower than in the upper extremities; the trunk can be flexed on the thighs so that the head lies between the legs (closed jack-knife position). The knees also can be flexed with the hip abducted so that the feet are placed on the pubes. There is a tendency to club feet on both sides and recently a definite talipes equinovarus on the left; there are no contractures, localized atrophies, hypertrophy or pseudo-hypertrophy. Flexion and extension at the hips are absent; abduction and adduction are entirely absent in the left and nearly so in the right hip when the limbs are extended. When the knees and hips are flexed there is moderate power of adduction, better on the right. Dorsiflexion of the ankles is slow and weak but movement of the toes is normal.

In rising from a reclining position, the patient rolls toward the right, supporting herself on the elbow and pushing it upward so as to raise the right shoulder; then with a jerk of the left shoulder she throws the left hand across, the trunk to the floor on the opposite side. The palm of the right hand is firmly planted on the floor by extending the right elbow; the trunk is gradually and with difficulty raised to the sitting position and the left arm is slowly withdrawn to its side. She then bends forward, takes hold of the legs just above the ankles and places the feet at the crotch so that the heels touch the buttocks. She throws her hands forward and to the right and stands on "all fours"; she then places her left hand on the left knee and, with a jerk, the right hand on the right knee and climbs up herself, jerking the head backward, the arms forward and the left leg outward to stand with a broad base.

The gait is broad-based and waddly. She looks downward and swings her arms as she steps, as though the arms assist in walking. The entire sole strikes the ground. The gait is like that of one walking on stilts; the trunk sways from side to side and the shoulders act as propellers. She walks unaided for about 30 to 40 feet. She drops to a sitting position without assistance. Walking is more difficult in cold or hot weather and in the later hours of the day. On retiring, the mother has to lift the patient's legs into bed and place them in extension. When a limb is lifted and released, it falls entirely without tonus.

The face is somewhat asymmetrical, but there is no facial palsy. The muscles of mastication are intact and deglutition is normal. The pupils are equal, regular, active to light and in accommodation. The left external rectus is paretic and there is a nystagmoid movement on inward excursion of that eye. When an object is placed directly in front of the left eye, the right eye ball rolls inward; there is no diplopia. The fundi are normal. Vision, hearing, smell and taste are normal. The tongue protrudes and opposes well, without deviation or tremor. Speech is distinct and of even tone; the voice is low. The teeth and tonsils are in good condition.

The superficial reflexes are normal and equal on the two sides. The deep reflexes in the upper extremities are normal and equal; the jaw jerk is present; the knee and Achilles jerks are absent. Muscle and joint sense are normal in the upper extremities and apparently also in the lower. The vibration sense in the lower extremities is faulty. There is no ataxia of station (Romberg test). Sensation in all phases is not impaired except in the legs where appreciation of cold is diminished.

Electrical tests show marked reduction in the reaction to both currents, especially in the legs, more so in the left; there is no reaction of degeneration. The facial muscles and those of the neck react promptly to galvanism and slowly to a faradic current of moderate strength. In the lower extremities a strong faradic current is borne without flinching.

The hair is coarse and dry, the skin soft and perspiration is scant. The legs are cyanotic and cold, and the integument is thick and tense. The nails are apparently normal. Sores heal very slowly and leave brown pigmentation.

Comment.—This case is a combination of myopathy with amyotonia, the latter predominating. That the onset was after birth is certain. She walked very well for more than a year. She was two and a half years old, when she was noticed to be limping, just after a severe attack of whooping cough. The symptoms indicative of amyotonia are: apathetic facies without paralysis; extreme hypotonia at all joints except wrists and ankles; winged scapulas; involvement of lower extremities; ability to perform normal purposive move-

ments, though feebly. There are no paralysis, atrophy, reaction of degeneration nor muscular twitching. The myopathy is indicated by: gradual onset after learning to walk; myoclonic movements; the gait; the manner of rising from the floor; club feet; and vasomotor changes in the legs.

The mother's obstetrical history should be considered as an etiologic factor. There is certainly marked reproductive disability. The first miscarriage may be said to have been accidental. All the other obstetrical mishaps occurred after the father had an attack of typhoid fever. Guillain observed three cases of myopathy following typhoid fever, but there is no case on record to show that the disease may be due to typhoid fever in either parent.

DISCUSSION

DR. JAMES C. GILL: Was the left external rectus paralyzed? Did the eye always remain turned in?

DR. YUDELSON: Under ordinary conditions the eye remains turned in, but it can turn out. It does not go beyond the median line except with great effort, and then the nystagmoid movements occur. I consider this a paresis of the external rectus.

DR. GILL: This is an interesting case, but I see no reason for classifying it as amyotonia congenita. It closely resembles the infantile myopathies commonly observed. With our present knowledge it seems to me confusing to combine these two classifications. I can see no difference between the description given by Dr. Yudelton and many myopathies. They do not all correspond to a type; some are rapid while others are slow in their progress and we meet all grades of this group of disorders.

DR. YUDELSON: The title chosen was given to avoid applying the name amyotonia congenita, and yet to show that many of the symptoms of both disorders may occur in one case. The symptoms in this case are those of a myopathy; hypotonia is also marked, though, as the history indicates, this case is not one of amyotonia congenita.

THE FACTOR OF DETERIORATION IN CHILDREN SHOWING
BEHAVIOR DIFFICULTIES FOLLOWING EPIDEMIC ENCEPHALITIS. DR. MANDEL SHERMAN and BERT I. BEVERLY (by invitation).

This report will be published in full in a later issue of the ARCHIVES.

Book Reviews

CONCERNING PSYCHANALYSIS. Introductory presentation by RUDOLF ALLERS in Vienna with following discussion in the Society for Applied Psychopathology and Psychology in Vienna. (Summer Semester, 1920.) Edited by Prof. Dr. med. ERWIN STRANSKY, first presiding officer, and Dr. med. et jur. BERNHARD DATTNER, first secretary. No. 16 of Contributions in Neurology, Psychiatry, Psychology, and Their Allied Fields; supplements to the *Monatschrift für Psychiatrie und Neurologie*. Price, 40 marks. Pp. 119. Berlin: S. Karger, 1922.

We deal here with an unusual contribution, a criticism of psychoanalysis (45 pages) by the neurophysiologist and psychologist Rudolf Allers, and about seventy-five pages of discussion by fourteen members of the society. It is a remarkable exhibit of the capacity of presentation, and the acumen and alertness of the speakers of a group made up of critics, friends and neutrals among Vienna psychologists and psychopathologists. The chief interest of the book lies in this fact that psychoanalysis is here discussed from various widely divergent points of view including orthodox psychoanalysis, academic clinical psychiatry, and phenomenologic psychology. A full review was therefore deemed desirable and essential, but proved decidedly difficult. An attempt was made to single out the special topics touched in the discussion so as to get into the smallest space and with maximal fairness a summary of the various opinions. Yet the reviewers fear that perhaps nothing but a perusal of the original could give a fair idea.

Allers and Schilder and most of the speakers use or refer to a cut-and-dried logic and formula very different from the psychodynamic and pragmatic formulations on this side of the Atlantic. They measure the product of Freud's life work by the criteria of another system of thought, instead of bringing both systems closer to the fundamentals. It might look like an unfair self-sufficiency to claim that there is a superabundance of chaff in such a procedure; but it certainly would be wiser to make a review of the whole phenomenologic and modern epistemologic approach to clear the atmosphere before taking up an inevitably complex problem like psychoanalysis with an alien system of logic; provided, of course, that one feels that enough would come from such a task. The influence of Husserl makes itself strangely felt in German writings, and yet there is very little recognition in Anglo-Saxon philosophic and psychologic literature of any acceptance and use of this line of thought. The spirit of William James and John Dewey and the formulations of psychobiology follow essentially different lines with far more respect for pragmatic fundamentals. The psychiatric applications of Husserl's ideas are to be found in Jaspers. Too much of the discussion is an attempt to measure Freud with the rules of the special type of game. By eliminating the application of Freudian theory in treatment, the discussion eliminates the essentials of method and the source of most of the data, and thereby it loses its natural interest. Allers, on purpose, keeps his discussion on a definitely abstract level—to our minds unfortunately so because the whole is so very empirical and conjectural at the same time.

Allers limits the discussion to psychoanalysis as a theory and method of research, shutting out the therapeutic aspect, as its results might not be any

proof of the theory. Since there is no concise and complete systematic presentation of the doctrine, Allers offers his own formulation of the theory.

He sees in psychoanalysis: (1) a method of investigation to recognize mental connections; (2) a theoretical conception of the nature of the soul-life of man; (3) a therapeutic procedure (not to be discussed); and (4) a claim of giving fundamental explanations of all possible domains of human life—the science of religion and art, philosophy, pedagogy, sociology, and ethnology. He does not depict it in its historical evolution, but as a finished claim or theory of psychologic organization as found in Freud's writings. He gives a very full and lucid outline of his conception of the theoretical foundation of psychoanalysis. This outline is rather cleverly formulated to arouse a host of questions, factual and theoretical. But Allers proceeds directly to the discussion of *psychoanalysis as method*, which is essentially that of free associations (reporting immediately every rising thought, without expurgations, critique, or shame), the watching for any gaps, and evidences of resistance with the light it throws on the charge of affect and the importance of the topic and the actual causal and intelligible connections. Allers refers to the assumption of "typical symbolizations" and "typical contents" of the unconscious existing in all human beings (such as the infantile sexuality, the incest problem, etc.). The unconscious is made up of phylogenetically and ontogenetically old material; its thinking is archaic; it realizes itself in pictures, not in words or thoughts; it keeps its affects and therewith its dynamic potency in the face of the barrier of censorship against entrance into consciousness. When the previously unconscious material becomes conscious, it is usually, but not always, recognized as the person's own.

Psychoanalysis claims to be an empirical science, Allers continues. It postulates a strict causal determinism (in contrast to Bergson's assumption of complete or partial freedom); psychoanalysis is interested largely in causal explanation, not in the phenomenologic accounting (so strongly emphasized by the followers of Husserl, Jaspers, etc.). The theory works with energetic concepts, and assigns a rationalistic nature with reasons and concepts and thinking even, to the unconscious.

This factual part of Allers' address is not specifically challenged in the discussion; but more so his excluding the practical experience.

While admitting that psychoanalysis has brought out a number of valuable discoveries, Allers turns from this objective account to his criticism of the *method* employed. He claims that he need not go to the trouble of actually employing the psychoanalytic method for the control of the facts if he can show that the method, for any reasons whatsoever, either is bound to furnish certain special results or is never able to justify certain others. Correct results might be obtained, not because of, but in spite of, the psychoanalytic technic. Therapeutic results, for instance, should not be taken as indubitable proof of the soundness of a method or theory.

The method is that of free association, the fundamental rule of communicating every arousal even if disagreeable or apparently irrelevant. Where gaps occur or the flow stops, the analyzer and the analyzand together have to overcome the resistance, and thereby they will bring out significant material from the unconscious. Allers sees in this a fatal *petitio principii* (p. 17). The mere fact of interruption of the free flow does not prove the existence of resistance and repression, unless other facts can be adduced as proof. In reality the hypothesis of repression anteceded the method of free association. It originated during the stage of the cathartic procedure. Neither the claim

of a resistance in the delay of the flow of association nor the causal significance of what is brought out by the effort of filling the break or gap is self-evident, and the conviction of its importance appears smuggled in by the introduction of the *concept of determination* which has at least four roots: it may point to a logical connection by sense or meaning, or to an associative relation of coexistence with or without relation by sense, or it may hang together emotionally, or it may be causal. Does the presence of symbols indicate causal relations? There may be a relation by meaning, but if an unknown quantity in an equation is designated by x , or if children use a piece of wood as a doll, we cannot say that the unknown *causes* the x , or the doll the piece of wood. The assumption of resistance and of a causal relation should not be assumed to exist without proof, merely on ground of the theory; and the theory cannot be properly based on such an assumption—hence a vicious circle. Further, the fact that “free association” is after all starting from components of a dream, or even from actual gaps in the dream, and has analysis as its goal, makes the association far from free. Moreover, there is no real end to the chain; each chain should properly include the totality of the psyche—correctly enough, but to the discomfiture of a theory of causation: each phenomenon would have the indivisible whole as its cause, and there would not be any distinctive difference in the causal importance of the different phenomena. The string of association is considered sufficient under the criteria of plausibility, of convergence upon a central point, and, when practicable, of the acceptance by the analyzand (the person analyzed). The analysis is sufficient if it gives a plausible picture of the latent thoughts, and this is based on the assumptions that a dream *does* represent an intelligible nexus of latent thoughts—which is a third *petitio principii*. Historically the theory arose in this way: the disappearance of the symptoms seemed to justify the conclusion that the disclosed material was causally effective and also determined the sense. “Can this be applied to the dream? Does the dream element disappear when the unconscious elements represented by it become conscious? There can be no disappearance because it is no longer present.” (This passage is a fair illustration of the sophistic method of Allers.)

Allers can therefore sum up his logical argument as follows (p. 21): The claim that the method leads to the conclusion and inferences of psychoanalysis rests on three vicious circles: that of the resistance, that of the causal determination, and that of the sense. It presupposes the whole of the theory which it claims to prove in each of its parts. The facts are: the strings of associations; and the occurrence of gaps. Everything else is interpretation resting on the theory to be proved. Yet the facts resolve themselves to nothing but this: Sometimes one associates and sometimes one does not—and “this finishes psychoanalysis methodically.” But there are other very startling aspects which Allers does not want to leave undiscussed.

The so-called laws of dream formation are merely laws of possible connection by sense, but not of causation. They largely coincide with the laws of association. Association by contrast may lead us to think of “secret” in a dream where there appear many persons; the latent element “secret” does not prove that the secret is the cause of the specially great number of dramatic personae. The association by contrast might very well have a root deeper than that of the meaning or sense of words; we know something like it in the phenomena of the negative after-image and in the phenomena of contrast. Allers admits, however, his reluctance in using physiologic analogies for the elucidation of psychological facts.

The occurrence of sexual and infantile material in the chains of association need not surprise one, since in the mental life everything somehow is connected with everything else, but by no means necessarily causally connected.

Allers claims justly that as long as psychoanalysis calls itself an empirical science, it should use controls. The invention of dreams seems to have been tried by Schroetter, but inadequately. Allers himself recommends the exposing of a picture and treating the impression as a dream with subsequent analysis—actually with very much the same result as with dreams, bringing out incestuous complexes, etc. Allers clearly is not inclined to look upon unconscious determiners as causes. He attributes the neglect of controls to the fact that the analytical conclusions were not obtained by the analytic method, but rather the method from the conclusions or intuitions.

He next turns against the interpretations and their (pathographic) application to dead personages in whom control is excluded. Not only the authors whom Freud calls irresponsible, but Freud himself indulges in the interpretation of "typical symbols." As to the sexual orientation of the interpretation, Allers admits that ethnology and folk-lore and ceremonials show that sexual associations do exist and are frequent, but not that they must be so of necessity. He quotes their occurrence in some ancient dream interpretations in an old Talmud tract. The generalization that hallucinations and delusions must be related to sex complexes he attributes to the diallelon (definition in a circle) of determination. With the discovery and appeal to "the unconscious yes" and the claim that there is no "unconscious no" the analyzer can always overrule the analyzand.

From the dissection and disposal of the "method," Allers passes to the discussion of the *concept of the unconscious* (p. 24). Disregarding those who object to the term, he accepts as recognized even outside of psychoanalysis the concept of an unconscious and the terms "unconscious mental life" and "unconscious thinking"—as a description of a certain type of fact, or as an hypothetical formula, a consideration of "as if" there were such a thing. He does, however, question some points concerning the special structure and mode of functioning assigned to the unconscious by psychoanalysis. He thinks little of the "metapsychologic" claim that the unconscious contains only concepts or images of things, whereas the conscious adds word concepts. This does not do justice to the phenomenology, i. e., the facts of consciousness as observed. We are supposed to assume that the unconscious contains what is repressed and unfit for, if not incapable of, consciousness, which has, however, a tendency to become conscious, either disfigured by the censorship or in its true form when the censorship is removed—in the cathartic procedure by hypnosis, or in free association through a force overcoming the resistance. But what is this force which is assumed to overcome the resistance? There evidently is another way, different from the overpowering of the censorship. Freud assumes for mental life something like the pattern of a reflex apparatus. The stimulus passes through several strata: a perceptive system, then memory systems, next the unconscious, the foreconscious, finally the conscious and the transformation into a motor act. Allers objects to the assumption of a close neuropsychologic parallelism, and the substitution of parts of the two systems for each other, and such expressions as the repression of old established association paths.

Between the unconscious and conscious the censorship is supposed to dominate, with selection and repression. If, however, the stimulus is regressive, from the conscious to the unconscious toward the perceptive spheres, there is

no need of overcoming the censorship (p. 26). The unconscious material then presents itself in the form of hallucinated memories. The process is the same as in remembering, in the search for a forgotten word. It is true, no explanation is offered as to how the images of the perceptive sphere reach consciousness. Allers sees in this a gap in the phenomenologic consideration, but one not limited to the psychoanalysts. If remembering is regression through several strata into the unconscious, one should expect that the memory too would rise primarily as hallucination or image. If one uses the expression hallucinatory, one evidently introduces a fundamental distinction between concept and percept—evidently a problem for the neglected phenomenology. In trying to remember the andante of the Fifth Symphony, one experiences what William James called an active gap. Allers notes a sense of what is wanted, over and outside of the various trials, until fragments or the whole memory comes up with a feeling that what is wanted is found. Similarly that which rises as image is not a representation of the non-perceptual idea in a conceptual form; one gropes for merely superficial features, because one finds oneself "incapable of holding on to the non-perceptual thought." As one of the many possible forms of remembering he mentions how Grillparzer, trying to remember a quarrel he had seen, recast it in terms of a chess game. There is no fixed rule of regression working only with the fact-concept or thing-concept and the word-concept.

Allers makes it appear as if psychoanalysis included in the unconscious the original drives, that which is repressed and incapable of consciousness, and probably also that which remains at any given moment as latent knowledge and latent "Einstellung" or set. It is only out of the unconscious that we can understand character-traits, peculiarities of the conduct of life and scientific and artistic productions.

One cannot help feeling that Allers takes us into a circus ring in which he rides the horse of a very elaborate and pedantic psychologic analysis, clad in the trappings of the psychologic vocabulary of detail observation similar to that of the Külpe or other introspectionist schools. Thus he suggests the working of the goal, the Aufgabe, where affects, but not contents, rise from the unconscious to influence the conscious.

Allers now (p. 30) turns to a second important part of the psychoanalytic doctrine, that of *sexuality*, which is contrasted with the ego-tendencies. The latter have received but little attention except perhaps in Freud's article "Jenseits des Lustprinzips." The modifications and expressions of sexuality figure as the causes of neuroses, psychoses, and normal phenomena. Libido, and that the libido of sexual nature, is made to include all psychic energy, or at least all that which serves the realization of the pleasure principle. Allers wants to call the pleasure derived from one's own body autohedonic and to distinguish between sexual and asexual tendencies. Similarity of the expressions should not be made to figure as identity. Thus a resemblance of religious ecstasy and of expressions of sexual experience does not prove that religious ecstasy is of sexual nature. Nor is sexual love *merely* sexual. The erotic component is only one of the most peripheral contributions of the central love act.

These fragmentary considerations concerning the concepts of the unconscious and of sexuality may not constitute as fundamental objections as were raised against the method. The method used and the results appear incompatible to Allers. Psychoanalysis works with a quantitative consideration of mental life, and he objects seriously to the psycho-energetic treatment of the

emotional phenomena, the concept of the "Affectbetrag" and of entropy, etc. The sum of affects (quantities of affect) is transferred from one concept or topic to another; converted into physical phenomena, or disposed of by "abreaction" or by "conversion." These are notions naïvely borrowed from physics. Allers objects seriously to the application of the category of quantity to mental facts. One does, of course, speak of "more love," of "stronger feelings," "greater hatred," and even Kant speaks of intensities in the anticipation of perception, and Fechner of quantitatively equal increase of sensation. Allers claims that the concept of quantity holds only in the physical world. If hatred or love or a sensation is "stronger," it is not merely stronger but fundamentally and essentially different from a weaker one. In ordinary pragmatism the term may be tempting and permissible, and also the passing from the purely mental realm of immediate experience to the spatial-quantitative realm of external reality. On this point even academic psychology should not blunder any longer. He quotes Fritz Mauthner's condemnation of the use of the energy concept in the mental sphere and in memory which has no spatial relations and furnishes no unitary measure.

Psychoanalysis and psychophysics fatally neglect the phenomenalist orientation which treats mental life as an essentially indivisible whole, and involve themselves in all the errors of a dynamic association psychology. What can the energy be which is supposed to pass from sexual excitement into anxiety? How can one really speak of "sublimation?" The moral and social qualities of man are supposed to arise by sublimation from sexual energy or energy attached to sexuality. Psychoanalysis assumes a primary repressive force. Where does it come from? Probably these difficulties have led to the opposition of the ego and sex tendencies. Allers sees in this not merely a lack of completion of the doctrine, but a deeply rooted fundamental flaw.

Thus Allers arrives at the following summary (p. 39): The psychoanalytic theory cannot be derived from the empirical "psychoanalytic" material unless one presupposes the theory. Three vicious circles appear: the *petitio principii* involved in the "resistance," in "determination," and the "intelligible connections." Hence it should not be supposed that the justification of the theory could be found in a definite set of fundamental facts; other categories would have to be found. Moreover, it follows that a control of the claims of psychoanalysis with its own methods is absolutely devoid of purpose and sense. The system has inner contradictions and is based on an entirely untenable peculiar psycho-energetic fundamental conception. With all this, we recognize the fact that we owe to psychoanalysis a great number of intuitions which strike one at once as new, valuable, and dependable, partly factual and partly in the way the problems are formulated.

Allers doubts that the categories of individual life can properly be applied to the group and totality as the psychoanalysts do with regard to the social and ethnologic phenomena.

He next asks (p. 42) how such a structure could have been built up by a man of Freud's mental capacity, apparently based on logical mistakes, and how the doctrine could acquire such a following.

Psychoanalysis arose at a time when the collapse of the natural history materialism and rationalism, with its tendency to "explain," began to lead over to the desire to "understand" connections (the passage from causalism to conditionalism). Symptoms called for an understanding out of the individual structure of the patient. But after all the attempt succumbed to a rationalistic tendency. Psychoanalysis had started out to understand a living

entity, but ended in explanations of single moments. This fundamental tendency and the actual performance give rise to an antinomy which explains all the defects criticized. A fundamentally understanding procedure and an exquisitely rationalistic theory are incompatible. The procedure bent on understanding gives psychoanalysis its intuitions and its therapeutic results, but not its explaining theory and method based on causality.

It seems convenient to present the discussion of Allers' lecture by arranging the various contributions according to the essential topics rather than by following the original sequence of the different speakers.

With regard to Allers' outline of the discussion, Schilder points out that, regarded from a historical point of view, psychoanalysis started on empirical-inductive, not on speculative-deductive lines. It is doubtful whether a formal-logical approach is the right form of criticism for psychoanalysis. He is convinced, however, after having discussed the more important topics of Allers, that psychoanalysis stands well also the test of a formal logical critique. Fröschels contradicts Schilder's contention of a purely empirical science; science begins where special categories reign. Federn regrets that Allers did not include the therapeutic use of psychoanalysis; it is not a question of the success of this therapy, but of the advantage of actual observation and research with patients, which brings out the psychic connections much more clearly than observation of normal people, or simple reading of psychoanalytic literature, which is the case with Allers.

Pappenheim feels that the "imminent criticism" of Allers is not convincing. A refutation of the psychoanalytic method in the way in which Allers approached this task is not possible. A discussion and criticism of the results of Freud's doctrine would have been more profitable than his methodologic examination. Roffenstein agrees with Allers that the theoretical foundation of the analytic method is insufficient; on the other hand he quotes Bleuler's account of the practical efficiency of the method in opening up facts which no other method would have brought out; and he warns against dismissing the method merely because the theoretical deductions and interpretations may as yet be inadequate.

The proof that the psychoanalytic method as such leads to correct and dependable results is according to Federn threefold:

1. The occurrence of the same contents, in the child, in primitive man, and in the psychotic; this was not the basis of the psychoanalytic discoveries, but constitutes its later confirmation. Roffenstein later denies that there exists a confirmation of the psychoanalytic proposition by the psychology of primitive man. Freud, in his "Totem and Taboo," has not found the neurotic mechanisms in primitive man as independent parallel happenings, but he has reinterpreted the psychology of primitive man by his own method.

2. The feeling of the analyzed himself that a correct connection has been disclosed, with an experience of definite inner relief.

3. The change in symptoms in the analysis of a neurotic patient. For Pappenheim also the empirical fact of the mode of disappearance of symptoms is a more important point for the justification of the analytic procedure than any general "immanent" criticism.

With regard to the method of free association and the significance of resistance, Schilder discusses the fundamental importance of the fact that our flow of associations is dominated by what Ach called "determining tendencies"; these tendencies regulate our thinking as "latent determinations," of which the individual need not be conscious. This is one of the kind of facts, first discovered by Freud, which constitute the real foundation of psychoanalysis, which

aims to formulate observations of a latent character as well as the obvious data. Gaps in the free flow of associations must therefore be of the greatest significance, and the formulations of censorship and resistance express this fact in an excellent way. Further, the method of free association is not meant to lead only to a knowledge of these underlying tendencies, but also to a knowledge of specific experiences from which a certain set of associations took its start. Federn charges Allers with a wrong view of the psychoanalytic method. Nobody can deny "resistance" who has ever experienced it as analyst. Resistance (or we might add "the facts for which this term is used"), is not an assumption, but an observed experience, fully confirmed by Jung's association experiments. He gives a short abstract of an analysis showing the evidently strong affective outbursts after the overcoming of the resistance, which manifested itself in the case in various very suggestive ways. He concludes his remarks by expressing the wish that critics of psychoanalysis may turn their attention not always to the basis of the method, but to those results of the method which are still uncertain, questions which he feels can only be solved by systematic psychoanalytic work, and not by theoretical negation. Pappenheim points out that the existence of the resistance is not assumed merely from the formation of a gap in the sequence of free association, as Allers had contended, but that there are a number of other phenomena which indicate the resistance, e. g., mimic movements, which repeat themselves sometimes in a surprising way when the same topic is touched. He quotes Bleuler, who maintains that the actual blockings are only "a small and quite dispensable part of the foundations of our conclusions." According to Hitschmann, only he can understand that a gap in the flow of associations indicates a resistance who has actually seen it *in statu nascendi* in the patient.

The question of the causal significance of the material gained by psychoanalysis is discussed by Schilder in a very interesting, though not altogether convincing way; he points to the aim of a true psychoanalysis to distinguish between "understandable connections" and causal connections in each individual case. His chief argument, however, seems to be that, after all, the original fact from which psychoanalysis started is the posthypnotic suggestion, where the conscious "motif" and the undoubtedly causal "motif," such as, for instance, the hypnotic suggestion, can be clearly distinguished. (It must be remembered, however, that historically, the starting point of the psychoanalytic doctrine was not posthypnotic suggestion, but the removal of amnesia, as practiced by Bernheim in Nancy. Freud says himself: "This astonishing and instructive experiment was my model.") Schilder also does not seem to do justice to the fact that Freud changed his attitude with regard to the causal significance of a psychic trauma. Freud, it is true, came to a new formulation on strictly empirical grounds, but this change is of vital importance with regard to the theory of psychic determination. Hitschmann states that psychoanalysis does not maintain that every psychic content is strictly causal and in every case only psychically determined. What psychoanalysis maintains is that in many cases psychic determination can be proved to a far greater extent than had been assumed before. Roffenstein, who discusses the problem of causal relationship at some length, lays stress on the fact that the form in which the causality concept appears in psychoanalysis is that form in which it has appeared first in the history of human knowledge, and has become the paradigm of all later causality concepts, viz., the expression of affects, of instincts, of volitional energy (p. 73). Allers contradicts this argument in his conclusion (p. 107), because we experience in our will first the fact of

being directed toward the realization of an imagined situation and therewith presuppose the possible connection between cause and effect.

With regard to the question of an intelligible sense of the dream, Pappenheim contradicts strongly Allers' view that the logical justification to interrupt an analysis at a point where one finds a possibility of construing a sense in the associated material, should rest on a *petitio principii*. He feels that there is no better example of a logical induction than when we draw conclusions from the disappearance of a symptom as to its causal connections with other symptoms. Hitschmann wants to confess to Allers that not every dream can be interpreted, and that we cannot always be sure whether we deal with a symbol or not. On the other hand he affirms that the physician interrupts the chain of association whenever he finds a crucial point indicated by a series of convergent associations and when he has got the conviction of a cogent interpretation. He thus admits the enormous freedom of interpretation in the analyst.

Concerning the question whether the results were obtained by the method or by intuition, Federn contends that a man must be "blind to the ways of research," if he really believes that Freud could have found a single one of the results of his doctrine without the psychoanalytic method. Hitschmann believes he has found a proof for his view that Freud has obtained his results inductively by the psychoanalytic method and not intuitively as Allers affirmed, in the fact that Freud's results were far too improbable for an intuitive discovery. He accepts only the first idea, that the dream may have a sense, as intuitive, and also the first attempt to put symbols in the place of typically recurrent dream elements for which the dreamer did not bring forth further associations. Everything else was careful and laborious observation and reexamination. Roffenstein regards Allers' statement of a knowledge "despite" the method instead of "through" the method as an improbable hypothesis; a clear distinction between method and result is not possible in the face of the close interconnection of the method with the partial results continuously obtained and the verification by the same method by pupils of the most divergent mental constitutions. Whatever may have been achieved by intuition, the theory, just as in chemistry and physics, could not have been achieved entirely without the method.

The psycho-energetic viewpoint to which Allers takes objection, is defended by Schilder, who points out that we deal here with an obvious hypothesis, independent of the fundamental doctrines of psychoanalysis. Neumann, who represents the philosophically oriented psychology and phenomenology, objects to the psychoanalytic doctrine of "psycho-energetic transformation," e. g., the transformation of libido into anxiety, because among other reasons anxiety is an experience and experiences cannot be transformed, nor can they arise by transformation. It seems to him that the psychoanalytic theory does not distinguish sufficiently two different concepts of quantity (or rather of intensity), the intensity of experiences and the intensities of psychic energies.

Hitschmann thinks that control experiments to the interpretation of dreams are impossible and valueless, without, however, making his point quite convincing. Roffenstein on the other hand, urges that control experiments on a large systematic basis are necessary, especially to determine whether psychic productions, not produced by the patient, would give a sense in the analysis. In this case then, one could of course not speak of causality. He describes such a control experiment of Bleuler, where an artificial dream did not yield a sense on interpretation. Poetzl (p. 91) draws attention to his own control

experiments, which proved the validity of Freud's dream analysis. Dattner, who discusses the dream experiments by Scrötter, also thinks that control experiments, especially for the symbol dreams and interpretations, would be a very welcome support.

Roffenstein discusses in some detail the concept of the "Unconscious." He finds in this concept two different inner contradictions: (1) The assertion of the unconscious as something totally different from consciousness, as "a second empire," is not in harmony with the empirical unity of the whole personality, which demands a feeling of memory and confirmation for all psychic contents. (2) The assumption that we should claim as criterion an immediate empathic understanding (*empfindbares Verstehen*), as proposed by Schilder, with his appeal to Husserl's concept of intuition, is in conflict with the definition of the concept of the unconscious, which, as something totally different from consciousness, should not at all be immediately intelligible by the means of consciousness.

Schilder designates Allers' view as erroneous, that Freud sees everywhere only *sex*. Freud distinguishes between sex-instinct and ego tendencies, the concept of narcissistic libido being a sort of intermediate stage. It is true that the ego tendencies have not yet been elaborated as thoroughly as the sex instincts. Hitschmann also contradicts Allers' statement that, according to Freud, every pleasure is sexual pleasure, and every unconscious impulse is fundamentally sexual; he designates this as a distortion of the psychoanalytic view. He raises what seems to be an excellent point against Allers' reproach that psychoanalysis had originally altogether neglected the ego tendencies. He regards it as a matter of course that as psychoanalysis started from the study of the transference-neuroses, repression was postulated, and the existence of a repressing principle was taken for granted, and the attention turned to the repressed facts. He admits that the examination of the ego tendencies has not yet been completed. He repudiates "the polymorph-perverse baby," mentioned controversially by Allers; all that Freud said is: "It is instructive that the child can become so under the influence of seduction." Freud has concluded retrospectively from his analyses that there is a close connection, an identity between the early and the later unmistakable actions of the erogenous zones. Allers maintains against this, in his conclusion, that Freud does actually speak of a "polymorph-perverse baby" in a different place. Mattauschek contends that the numerous war experiences have convincingly disproved the exclusively sexual etiology of the neuroses. In order to obviate confusion, Roffenstein calls for an answer to the concrete question, not sufficiently heeded by psychoanalysis: what is the distinction between sexual joy and sexual instinct on the one hand, and non-sexual joy and non-sexual instinct on the other—introspectively as well as objectively—if psychoanalysis considers as inadequate all our former biologic, anatomic, physiologic, and psychologic points of distinction?

Two further topics, both intimately connected with each other, and of importance in any discussion on psychoanalysis, but outside of Allers' original outline of the topics to be discussed, are raised by Fröschels, viz., the problem of suggestion in psychoanalysis and the question whether catharsis alone can effect a cure. Fröschels maintains that it is not possible to cure a psychoneurotic patient by catharsis, by digging out, as it were, a pathogenic experience or psychogenic experiences, because too much of what happened after the pathogenic event has itself become later a psychoneurotic complex or part of a complex; the soul being always a unity, it is impossible to get

hold cathartically of a pathogenic experience unchanged and without leaving a residual. The psychoanalytic cure necessarily includes suggestion. Roffenstein, who feels that the mechanism of the "father transference" alone does not settle the question of suggestion, also considers suggestion an important factor in psychoanalysis, not only for the direction of the flow of associations, but also for the therapeutic success.

Hitschmann draws attention to the conception of transference, "one of the most important observations of analysis." He contends that Allers has unduly neglected this point and this is the reason why he (Allers) cannot understand how the resistance is overcome during an analytic cure, a result obtained by hypnosis in the cathartic cure.

Roffenstein brings a new point in the discussion, the problem whether dreams and neuroses are really only regressions, or whether they do not have a "prospective tendency" as the Swiss group and Adler maintain. He thinks that the latter view corresponds better with a biologic point of view, for it is unlikely that a reminiscence, in the sense of a phylogenetic and ontogenetic residual, can have such important effects.

Trebitsch raises a new point in the discussion. He finds the explanation of the gulf between psychoanalysis and "school psychology" in the existence of two different types of mentality, according to the prevalence of affirmation or negation from early childhood. The negated type, such as the Jew, finds a refuge from the negation by the environment in a precocious eroticism, and becomes sensitized in a manner unintelligible to those who did not grow up under negation (i. e., the dominant race). The psychoanalytic doctrine deals only with this specific disposition, and is not generally valid. Stransky regards this point as very noteworthy, but warns against final formulations, especially in view of the strongly racial composition of the stubborn Swiss group of psychoanalysts.

As gains of permanent value and "unconditionally acceptable," Allers mentions in his conclusion:

1. A better appreciation of sexuality, especially infantile sexuality.
2. The recognition of personality as a unit.
3. The existence of different psychic layers and recognition of the mechanisms of repression, condensation, and displacement.
4. The recognition of symbolism, restricted to a non-causal relationship; and of
5. Sublimation, also with certain theoretical reservations.

Allers seems to agree in his conclusion with Neumann who sums up his point of view in his contribution to the discussion, that the discovered facts will remain, but "very little of the theoretical substructure." Though this seems to be an extreme statement of a critical attitude toward psychoanalysis, the book certainly is stimulating along this line of criticism.

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DIE EPIDEMISCHE ENCEPHALITIS. PROF. FELIX STERN, Physician-in-Chief of the University Clinic for Mental and Nervous Diseases, Göttingen. Paper. Price, \$2.80. Pp. 228. Berlin, Julius Springer, 1922.

This volume is the thirtieth monograph published under the auspices of the *Zeitschrift für die gesamte Neurologie und Psychiatrie*. It aims to cover in detail our existent knowledge of epidemic encephalitis, regarded as a clinical and pathologic entity. This is no light task, since, as the author states in the preface, no other subject in neurology, not excluding the voluminous war literature, has offered so many problems of pathologic and etiologic interest as has the pandemic of encephalitis. In spite of the fact that the first adequate description of the "new disease" by Economo was no longer ago than 1917, Stern gives in an appended bibliography a list of 372 references, about a third of those collected by the recent Report of the English Ministry of Public Health. The monograph is based on a personal study of 105 clinical cases and nine post-mortem examinations, together with a general review of the literature.

The subject-matter is divided into seven main headings as follows: (1) Clinical aspects; (2) Pathologic anatomy; (3) Epidemiology; (4) Pathogenesis; (5) Genesis of the somnolence; (6) Differential diagnosis; (7) Therapy. The first of these topics is discussed in great detail, taking more than half of the book in its exposition. This is an admirable statement of the manifold forms in which the disease may manifest itself, with constant text references to the work of other men and their opinions. The pathologic anatomy is treated in much more summary fashion in the space of twelve pages. It is, however, a good résumé of our knowledge, emphasizing the slight macroscopic changes, the non-hemorrhagic character of the lesions, the perivascular infiltration and blood-vessel changes, the slight tendency to softening, the predilection for certain areas, and the residual changes in the nerve tissue. Considering the epidemiology, the author does not question the existence of encephalitis of similar character previous to the present outbreak but there is no evidence that it has ever attained such proportions; and certainly it has never before been adequately studied. Its relation to influenza is regarded as not yet definitely determined; it is pointed out that the time relation of the two diseases has been inconstant and that in many instances encephalitis has preceded or been prevalent coincidentally with influenza, rather than consistently following it. On the other hand, their occurrence often in close association demands recognition of the fact that there may be a more intimate relationship between them than is as yet demonstrable. This matter is discussed in some detail in the section on pathogenesis. The statement is made that pandemic gripe and epidemic encephalitis stand in an indirect relationship to each other; that the essential virus of encephalitis is a filterable poison (noxe) which is present in harmless form in the mouths of many individuals; that the activation of this harmless "noxe" may take place through the virus of gripe which may also be activated by other organisms, for example, pneumococci or streptococci. The claim of a diplostreptococcus, as originally suggested by Economo and von Weisner, and the researches of Loewe and Strauss, Levaditi, Harvier and others relative to a filterable virus as possible causative agents, are critically examined and without prejudice, and also without any very definite conclusions. The discussion of the genesis of the characteristic somnolence naturally offers no solution of that unique phenomenon, but is interesting reading, particularly through the attempt to show its relation to normal sleep on the one hand, and to various pathologic

disturbances of consciousness on the other. Stern does not share the skepticism of many clinicians and investigators that convalescent serum has not demonstrated its value. He is very definite in his statement that such serum used in the acute stage is distinctly beneficial and quotes certain personal cases to establish his contention. Treatment by this means he thinks should be recommended in preference to the various drugs and other means of relief which have been widely used.

In general the monograph is readable, comprehensive in its scope and will serve as a most useful epitome of progress in the study of a disease, many of the problems of which have hardly been formulated, much less solved. There is no index; a somewhat complete table of contents does not atone for this omission.

MULTIPLE SCLEROSIS (DISSEMINATED SCLEROSIS). An Investigation by the Association for Research in Nervous and Mental Diseases. Cloth. Price, \$3.75. Pp. 241. New York, Paul B. Hoeber, 1923.

Again we are indebted to the Association for Research in Nervous and Mental Diseases for a valuable and handy monograph. (The Association's volume on lethargic encephalitis was reviewed in these *ARCHIVES* 7:151, 1922.) In December, 1921, the Association devoted a two days' meeting to the subject of multiple sclerosis and this volume of some 240 pages is the result. It contains the substance of all of the papers presented and of the discussions—if the procedure of quizzing the author can be called a discussion. There were twenty papers by twenty-six authors. These papers were taken in hand by the editorial board (Drs. Dana, Jelliffe, Riley, Tilney and Timme), properly edited and arranged in the following groups:

- I. General and Historical Considerations.
- II. General and Special Symptomatology.
- III. Differential Diagnosis, Course and Treatment.
- IV. Pathology.

At the meeting, after a paper was presented there was no general discussion but the author was questioned *ad libitum* by members of the Association. Occasionally this almost amounted to scientific "grilling." In the volume, the interrogation follows each paper, in some instances occupying ten or twelve pages.

At the close of each group of papers, e. g. General and Special Symptomatology, the Commission, which is the jury of the Association, briefly gives its conclusions. Thus the reader has not only the benefit of the author's presentation but also of elaboration and defense of his thesis elicited by the quizzing and of the mature opinion of a selected body of competent neurologists.

While the contents in detail and manner of construction of this monograph are far from those of the textbook or ordinary systematic treatise, the entire subject is covered and some of the matter is scarcely to be found elsewhere.

The historical introduction by Dr. Walter Timme is brief but adequate. Statistical papers by Drs. Charles B. Davenport and Pearce Bailey present new and interesting matter on geographic distribution, race incidence and incidence in United States troops in the late war. General statistics of the disease relating to morbidity incidence, incidence of age, sex, nativity, occupation, civil status, etc., are presented by Dr. Israel S. Wechsler, his conclusions being based on a study of 1,970 records, 1,773 from the literature and

197 from New York hospitals. Dr. Charles L. Dana contributes a brief paper on Multiple Sclerosis and the Methods of Ecology, we believe the first consideration of this matter for medical men.

There follow about forty-six pages by different authors relating to general and special symptomatology with some twenty-eight pages of questions by the Commission and answers by the authors. It may be noted that this section contains the results of special serologic studies, work on the spinal fluid, the last word on bacteriology of multiple sclerosis and a presentation of the attitude of a psychoanalyst (Dr. Jelliffe) on Emotional and Psychological Factors in Multiple Sclerosis.

The work on the bacteriology of the disease, by Dr. Oscar Teague under the direction of Dr. Frederick Tilney, although uncompleted, is especially important. It has been more extensive and painstaking than any similar investigation up to the present time and quite negatives some rather startling statements of other workers affirming a specific organism.

Regarding "the probability that certain action patterns resulting from unconscious psychologic repressions and determining certain disturbances in the vascular distribution which result in the plaque formation of multiple sclerosis," the conclusion of the Commission may be quoted. "At present the Commission feels that it must accept this proposal merely as a suggestion which, in the absence of confirmatory observations and further proof, has not yet been established on a sufficiently firm basis to determine its validity."

In the section on Pathology the paper by Dr. Hassin contains what are probably the best illustrations of the histology of multiple sclerosis yet published and Dr. Joshua H. Leiner presents the latest work on the axis cylinder in this disease. (The illustrations of these two papers are reproduced from these ARCHIVES 7:589, 1922; and 7:608, 1922.) The difficulties of correlating the location of the lesions with the symptoms in individual cases are well discussed by Dr. E. W. Taylor, with illustrative cases.

A classified bibliography of about 600 titles closes the volume. That there is no index is matter for regret.

THE CONSTITUTIONAL FACTORS IN DEMENTIA PRAECOX WITH PARTICULAR ATTENTION TO THE CIRCULATORY SYSTEM AND TO SOME OF THE ENDOCRINE GLANDS. By NOLAN D. C. LEWIS, M.D. Nervous and Mental Disease Monograph Series No. 35. Paper. Price \$3 Net. Pp. 134 with illustrations. New York: Nervous and Mental Disease Publishing Company, 1923.

The object of this study was to discover a basic constitutional factor in dementia praecox. The author distinguishes sharply between the hebephrenic, catatonic, and paranoid forms and regards the last as outside the province of true dementia praecox. The material was selected from the records of 4,400 postmortem examinations made at St. Elizabeth's Hospital and, after those instances in which the diagnosis was not clear and those in which there was a question of associated organic brain disease were excluded, there remained 601 cases of undoubted hebephrenia or catatonia. The incidence of pulmonary tuberculosis was so high that it became necessary to separate the material into eight groups: white males with tuberculosis, colored males with tuberculosis, white females with tuberculosis, colored females with tuberculosis, white males without tuberculosis, colored males without tuberculosis, white females

without tuberculosis, and colored females without tuberculosis. The following table is of considerable interest and gives the average heart weight in grams in each of the eight divisions.

Group	No. of Cases	Average Heart Weight
1	315	262.4
2	73	261
3	34	240
4	45	221
5	88	277
6	17	303
7	10	222
8	19	250

The average for the entire 601 cases, which was 254.4 gm. stands in striking contrast to the cardiac weights in arteriosclerotic dementia (399 gm.), senile dementia (362 gm.), parenchymatous neurosyphilis (281 gm.), manic-depressive (361 gm.), mesoblastic cerebral syphilis (331 gm.), epilepsy (366 gm.), and paranoid state (395 gm.). It will be noted that general paralysis is the only brain disease in which the low dementia praecox average is approached.

The monograph includes excellent detailed reports of the necropsy findings in twenty-two cases which will repay careful perusal. The chief items of interest may be referred to the aplastic circulatory system and the disorders of the thyroid, adrenal and gonads, with occasional involvement of the pituitary and mammary glands. The heart muscle is originally insufficient and the lumen of the aorta universally small—barely large enough to admit the first finger. Furthermore, the cardiac muscle lacks the capacity to hypertrophy, even in the presence of a valvular lesion. Microscopically, there is a lessened tendency for connective tissue replacement. The aortic walls are small, thin, smooth, and over-elastic and, since the main branches of the aorta likewise show aplasia, the assumption is that this is an index of the condition of the entire circulatory system. This corresponds to the circulatory aspects of the so-called status thymolymphaticus syndrome. The prominence of tuberculosis is presumably favored by the lymphostasis which exists on the following basis: "In most instances of dementia praecox constitution there is an aplastic or small circulatory system, and the lymphatic system, in order to account for intercellular spaces, must be large and wide. Since the widening of the bed of any stream produces slowing of the current, we have here perfect predisposing possibilities for the growth and advance of the lesions of tuberculosis without resorting to theories concerning prolonged confinement and lowered resistance, which factors also feature in other mental disorders not so often involved with tuberculosis. . . . The bacillus probably enters the hylus of the lung and passes along the peribronchial lymph channels to the periphery, and on account of the more sluggish lymph stream in the apices here produces its classical picture." The endocrine glands, particularly the thyroid, adrenals and gonads, revealed more or less constant gross and histologic departures from the normal and the author feels that the aplasias, atrophies, scleroses, and patchy hyperplasias are independent of the age of the individual, the duration of the psychosis, or the incidence of associated physical diseases and concludes that "they have suffered in the course of the development of the personality so that their respective functions are subsequently, imperfectly and aberrantly performed."

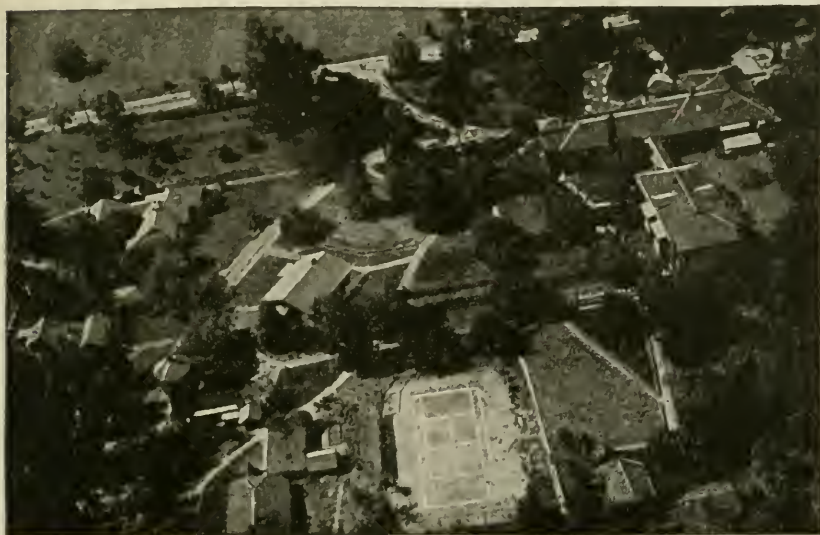
Naturally, the attempt to correlate the circulatory and ductless gland defects with the psychopathology and mental symptoms of schizophrenia implies a

considerable amount of supposition and yet the theory advanced is not without certain logical merit. The developmental shortcomings interfere with adequate response to a complicated environment and the organism regresses to a vegetative level where circulatory and endocrine adjustment is feasible. This would explain the preponderance of dementia praecox at puberty. The precipitating stress may be physical, chemical, psychic or social, in various combinations or in pure culture.

A minimum evaluation of Lewis' work is that it is a valuable contribution to the pathology of dementia praecox, the least understood of all the psychoses; the only permissible criticism is that there is not sufficient evidence to believe that the connection between somatic deficiencies and the mental picture is so simple and direct.

LAS ENCINAS

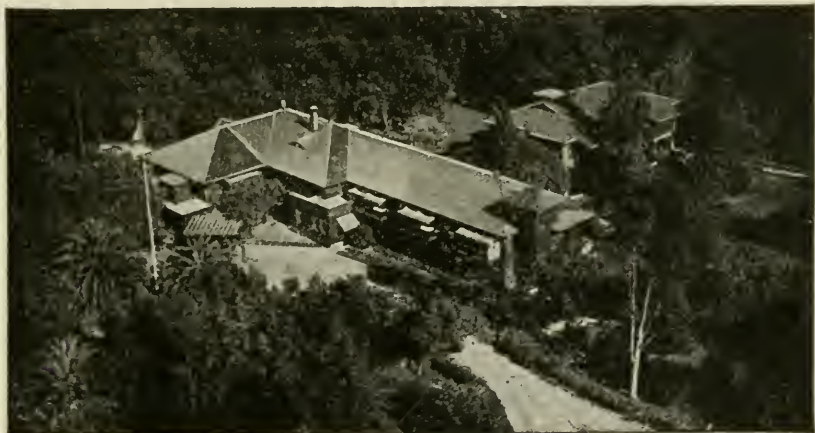
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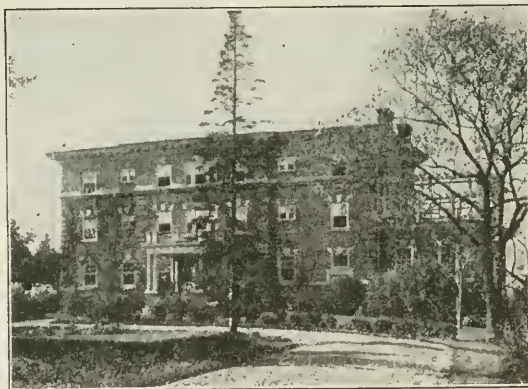
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THE MECHANISM AND THE FUNDAMENTAL CAUSE OF THE EPILEPSIES

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CONTENTS

- I. INTRODUCTION
- II. ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS
 - The State of Tonus and Its Dependence on the Suspension of Certain Cerebral Activities.
 - The Biologic Significance of Posture. The Normal Periods of Suspended Cerebral Activity.
 - Movement (clonic muscular contractions) as a Mode of Redistribution of Tonus.
 - The Relation of Muscular Activity to the Conscious State.
- III. THE NORMAL EPILEPTOID REACTION
 - The Epileptoid Reaction in Response to External Stimuli.
 1. The Stage of Disintegration of the Conscious State. The Aura.
 2. The Stage of Tonic Rigidity. The Paroxysmal Outcry. Anesthesia Associated with the Paroxysm.
 3. Clonic Movements.
 4. The Stage of Recovery.
 - The Biologic and Physiologic Significance of the Normal Epileptoid Reaction.
 - The Epileptoid Reaction Associated with Certain Functions.
- IV. THE PERIODICITY OF THE NORMAL EPILEPTOID REACTION
- V. EXPERIMENTS ON THE EFFECT OF CAFFEIN, BROMIDS AND PHENOBARBITAL ON THE EPILEPTOID REACTION AND THE EPILEPTIC PAROXYSM
 - Experiments on Reptiles.
 - Subjective Experiments.
 - The Effect of Caffein and Phenobarbital on the Epileptic Manifestations.
- VI. THE RECIPROCITY OF NERVE FUNCTIONS. THE IDENTITY OF THE EPILEPTIC PAROXYSM WITH THE NORMAL EPILEPTOID REACTION
- VII. SUMMARY

I. INTRODUCTION

The history of epileptology for the last hundred years is a record of sublime obstinacy in the face of increasing difficulties. With every addition of facts bearing on the disease, the hope of fathoming its etiology appeared more distant. In 1857 Brown-Séquard¹ was certain that he could produce epilepsy in animals by asphyxia or by injuries to the spinal cord. Sixty years later Silberstein,² having produced epileptiform attacks in animals by injuries to the brain and other portions of the nervous system, took pains to warn his readers that such injuries are not essential to the disease of epilepsy.

The production of movements simulating a localized convulsion by means of electrical or other excitation of certain areas of the cerebral cortex, has resulted in a wider knowledge of the anatomy and physiology of the central nervous system. Regarding epilepsy, however, the procedure has yielded little more information than the movements produced by stimulation of the spinal nerves. The application to the cortex, or the injection into the blood, of certain drugs, has taught the lesson that widely dissimilar substances, such as lead, the toxins of uremia, absinthe, or picrotoxin may, through their action on the central nervous system, produce convulsive movements. Yet this valuable lesson has furnished hardly any assistance to the understanding of the disease.

"If on the one side," wrote Reynolds³ some sixty years ago, "it is said that convulsions are induced by cerebral anemia; by eccentric irritation; by injury to the nervous centers inflicted by disease on the brain and on the spinal cord, or experimentally produced by lesions of the former or the latter; by blood disease and by general cachexia and debility, it must be said in reply that epilepsy exists when no one of these conditions is present; and the utility of their observation, *quoad* the pathology of epilepsy, is to show how, when they are not present, the special phenomena of that disease may be brought about; and how when they are present they are related to the central fact of epilepsy."

It must indeed be plain that if any disease exists both in the presence and in the absence of a certain condition, that condition, much as it may contribute to increase or to diminish its severity, cannot be considered as the causative factor of the disease; and every condition in connection with epilepsy thus far observed has been open to this objection. The very multiplicity and variety of factors lately described by

1. Brown-Séquard, E.: *Researches on Epilepsy*, 1857.

2. Silberstein, A.: *Experimentelle Untersuchungen über traumatische Epilepsie der Meerschweinchen*, J. f. Psychiat. u. Neurol. **22**:123, 1916.

3. Reynolds, J. R.: *Epilepsy*, 1861, p. 50.

Kraepelin⁴ as concomitant with different cases of epilepsy, is sufficient to convince anybody that such factors cannot but serve as contributories to the causation of the affection. Yet even if a constant factor concomitant with the symptom-complex of epilepsy were to be discovered, say of the nature of a chemical substance in the blood, proof would still be wanting that that factor is the cause of the disorder. Even if the application of such a substance to the cortex or its introduction into the blood of an animal regularly brought about a convulsion, we might still be justified in doubting its responsibility for the disease. For we know, on the one hand, that chemical substances exist in the normal animal body whose application to the cortex produces serious alterations of its vascular supply. An example of such a substance is epinephrin. On the other hand, we know that the effect of the introduction into the body of foreign substances is no measure of the effects of those substances on the organism to which they are native.

In certain diseases of known etiology, which are characterized by periodicity, we can discover times of definitely increased activity of the causative factor corresponding to the periodic manifestations of the disease. Malaria is an example of such a disease. In epilepsy on the contrary, the causative factors thus far assumed are continuous in their action, while the disease is definitely, though irregularly, periodic.

A review of the literature on the subject of epilepsy reveals a singular fact. In the eager search of a great number of investigators for isolated phenomena analogous to the several manifestations of the epileptic fit—in the search for examples of hallucinations, of reduced states of consciousness, of tonic and clonic spasms—the examples of such manifestations nearest at hand have been overlooked. It will be seen in the course of the present investigation that the entire train of these manifestations, though in less striking form, nevertheless in the same sequence and with the same periodicity as in epileptics, occurs also in all admittedly nonepileptic persons possessed of the average mental and physical states of health. It will appear, moreover, that this train of manifestations subserves a necessary biologic function in itself, and also that it is an indispensable part of a number of other physiologic activities of the animal organism.

A study of epilepsy from this standpoint has thrown light on a number of hitherto obscure facts.

The beneficial effect of such drugs as the bromids and phenobarbital in diminishing the frequency and the severity of the epileptic paroxysm, is an established fact. These drugs are nerve depressants and hypnotics. Their action in reducing the state of consciousness is

4. Kraepelin, Emil: Zur Epilepsiefrage. *Zeitschr. f. d. ges. Neurol. u. Psychiat.* 52:107, 1919.

well known. That the administration of drugs whose action is to reduce the state of consciousness should result in a diminution of the number and the severity of paroxysms, whose chief and frequently only characteristic is a reduction of the conscious state, appears to be a contradiction to the evidence of common experience. One of the results of the present investigation has been to reconcile this seeming contradiction.

Almost every investigator who has studied the disease, has found it necessary to call attention to an outstanding feature of its hereditary phases. An epileptic child may be born of nonepileptic antecedents. In such cases, however, it is generally found that the antecedents have been the subjects of neuropathy or psychopathy of one kind or another. Every investigator, too, has called attention to the fact that emotional shocks, infectious diseases, or traumatic injuries of any kind may produce the disease in a person whose nervous system deviates, however slightly, from the normal. The following two cases, from the series studied in the Neurological Division of the Vanderbilt Clinic, are illustrative of this point.

CASE 1.—Joseph C., aged 20, with a history of normal birth, infancy and childhood, experienced at 13 a severe illness of a few days' duration that was diagnosed as sunstroke. A week or two after recovery there occurred a major epileptic paroxysm. Other attacks of both major and minor epilepsy followed with increasing frequency and there was progressive mental deterioration. He has forgotten a large portion of what he learned at school before the age of 13, and he has acquired no new knowledge since. No history of epilepsy or insanity could be traced in the family. The mother had been a very "nervous" woman as long as she could remember.

Examination showed the reflexes to be normal. Speech was slow and sentences were poorly formed. He stammered occasionally. His ears were abnormally large and stood out prominently from the side of his head. The ring finger of the right hand was small, thin, and connected with a web to the middle finger. The space between the second and third toes was webbed.

CASE 2.—Charles S., aged 9, was reported to have had an easy birth without instrumental interference, though the head was abnormally large in proportion to the body. He was weak and sickly all through infancy. The first teeth appeared at 6 months. The first words were uttered at 2 years of age and he began to walk at the age of 4. At the age of 2 he had a severe febrile illness which began with a stiff neck and was of eight weeks' duration. At the age of 5 he had an attack of influenza that lasted a month. A week after recovery there was a paroxysm of major epilepsy, which has been repeated since on an average of once a week. No history of mental or physical defect in the family was elicited.

Examination showed the right side to be somewhat the weaker of the two and its patellar reflex slightly more pronounced. A positive Babinski sign was present on the same side. The abdominal reflexes were brisk. No abnormalities of the cranial nerves were found. There was no defect of coordination or equilibrium. The child's head was abnormally large. The left testis was undescended. Except for a rather precocious—a mannish—manner of conversation, there was no mental abnormality.

The congenital defect of the nervous system in the first case is inferred, though rather distantly, from the congenital physical defect of the webbed fingers and toes, and from a neuropathic antecedent. In the second, however, a congenital hydrocephalus bears a more proximate relation to a congenital abnormality of the nervous system. What bearing the undescended testis may have in this connection, we do not know. Whether the sign of Babinski, the exaggerated patellar reflex and the relative weakness of the right side date back to a hemiplegia at birth or to a meningitis at the age of 2, or to influenza at the age of 5, was not ascertained.

Both the congenital and the determining factors in the two cases are of widely different nature. They are examples of classes of cases to which every student of the disease has called attention. That different kinds of congenital defects, as well as different apparent determinants, should cause the appearance of the single symptom-complex of epilepsy is a point which has not been explained. It will appear from the present investigation that all such defects and determinants contribute to the enhancement of a normal reaction of a certain primitive order, to the extent of degrading it to the clinical picture of epilepsy.

Among the seemingly contradictory phenomena related to epilepsy is the fact that the same kind of stimulus is capable either of precipitating or aborting a paroxysm. Brown-Séquard¹ precipitated convulsive fits in his animals by peripheral irritation. Buzzard⁵ found that the irritation of a blister will prevent an epileptic attack. The constriction by a band of an extremity in which the convulsion begins is known to abort an oncoming seizure. Gowers⁶ and others speak of painful experiences as potent factors in precipitating a paroxysm. A loud noise, a whiff of ammonia, voluntary movements, and any number of most different conditions have been described as efficient in aborting a beginning attack; and it is the experience of every one who has made a study of the disease, that the same agencies are equally adequate in precipitating the paroxysms. Gowers,⁶ indeed, describes the case of a patient in whom attacks were first precipitated by certain involuntary movements and, later, by voluntary movements as well. From the viewpoint of the present study the seemingly inconstant and contradictory effects on the epileptic of these different stimuli becomes easily explicable.

Among the factors which mark the appearance of the first paroxysm, that of fright occupies a place of great prominence. "Of the three forms of emotion," writes Gowers,⁶ "fright takes the first place. . . .

5. Buzzard, Thomas: *Diseases of the Nervous System*, 1882, p. 425.

6. Gowers, Sir William R.: *Epilepsy and Other Chronic Convulsive Diseases*, 1901, pp. 25, 30 and 109.

The female sex is notoriously the more emotional, and accordingly the disease results from fright in a larger proportion of females than of males. . . . In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact." Throughout the literature on the subject no reason for so close a connection between fright and the epileptic paroxysm is to be found, and the rather vague assumption is everywhere made that strong emotions have the general effect of increasing the susceptibility to epilepsy of persons of a neuropathic disposition. A much more exact reason for the close relationship between fright and epilepsy has appeared in the course of the present study.

The subnormal mental and moral traits which characterize a number of epileptics have given rise to much conjecture, more mystification, and not a little of mystic and phantastic interpretation. They have been studied and described with an admirable degree of insight and thoroughness by Echeverria,⁷ Arndt,⁸ Binswanger,⁹ Spratling,¹⁰ Régis,¹¹ Clark,¹² and a number of others. The present investigation corroborates the findings of these authors. The reason assigned for the peculiarity of character on the basis of the present study, will be found, at its conclusion, to be comparatively simple and understandable.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

While it is generally admitted that the epileptic person, unconscious and rigid, or writhing in convulsions, is in a state similar to that of the decerebrate animal, writers on epilepsy continue, with a singular inconsistency, to speak of the epileptic motor phenomena in terms of cerebral cortical discharges. The obvious reason for this inconsistency is an effort to align two known facts with the epileptic convulsion. One fact has reference to the twitching movements produced by electrical or other excitation of the motor area of the cerebral cortex. The other is the fact that lesions of limited areas in or near the motor cortex are productive of localized or Jacksonian convulsions, the musculature involved corresponding to the area of the cortex which is known to preside over their contractions.

It will be shown later that the twitching movements produced by the experimental excitation of the cortex are most probably due to a

7. Echeverria, M. G.: *On Epilepsy*, 1870, p. 362.

8. Arndt, R.: *Lehrbuch der Psychiatrie*, 1883, p. 405.

9. Binswanger, Otto: *Die Epilepsie*, Nothnagel's System of Medicine, 12:313.

10. Spratling, W. P.: *Epilepsy*, 1904, pp. 287 and 438.

11. Régis, E.: *Précis de psychiatrie*, 1909, p. 881.

12. Clark, L. P.: *Clinical Studies in Epilepsy*, 1917.

release of motor function in certain groups of cells in the ventral portion of the spinal cord. The same principle holds good respecting the localized convulsions caused by localized lesions of the motor cortex. Far from being brought about by cortical "discharges," the results of the last twenty years' work in nerve physiology point indeed definitely to the conclusion that these convulsions are due rather to *the lack* of such discharges.

In order to rectify such misunderstandings as well as to afford a clearer view of the manner in which the epileptic attack originates in the particular biologic reaction disclosed by the present investigation, it becomes necessary to review, even though briefly, the facts relating to some of the nerve structures and functions most probably involved.

THE STATE OF TONUS AND ITS DEPENDENCE ON THE
SUSPENSION OF CERTAIN CEREBRAL ACTIVITIES

Clinicians noted many years ago that among the results of lesions which destroy a large portion of the cerebrum is a state of general muscular rigidity, usually manifested by tonic fits. Such a case was reported by Bastian¹³ in 1884. In the course of the next twenty years the increasing attention bestowed on this state of rigidity is evidenced by the greater detail of the descriptions. Thus, in the report of a case of intracranial aneurysm in 1904, Bruce and Drummond¹⁴ note that during the spasms "the limbs became rigid, the arms were extended in front of the body and rotated so that the hands were placed back to back." In the same year, Stewart and Holmes,¹⁵ in their treatise on the symptomatology of cerebellar disease, described cases exhibiting numerous phases of these abnormal muscular conditions with a degree of clearness that enabled the clinician to recognize them in previously obscure instances. Thus, in connection with a case of tumor of the under surface of the vermis and subsequent operative injury to the dentate nucleus, these authors describe a series of tonic fits in which "both arms were rigid and resistant to passive movement. . . . They were adducted to the sides, extended at the elbows and wrists, while the fingers were flexed at all the joints and covered the thumb."

While clinicians continued to study such cases with increasing attention, experimental physiology was locating the center of muscular rigidity and elucidating its mechanism. The striking phenomenon of extensor rigidity of the decerebrate animal demanded an explanation,

13. Bastian, H. C.: A Case of Apoplexy in a Boy Aged 15, Trans. Clin. Soc. **17**:21, 1884.

14. Bruce, Alexander, and Drummond, W. B.: A Case of Intracranial Aneurism in a Young Subject, Rev. Neurol. & Psychiat. **2**:737, 1904.

15. Stewart, T. G., and Holmes, Gordon: Symptomatology of Cerebellar Tumours, Brain, **27**:522, 1904.

and efforts were made to discover the pathways which, in the normal state of the animal, maintain its musculature in a moderate state of tonus. In 1897, Löwenthal and Horsley,¹⁶ gave the result of their observations on the relation of the cerebellar to the cerebral structures as follows: "When both cerebral hemispheres were removed and, as a result, active extension tonus of the limbs was obtained, excitation (faradic) of the upper surface of the cerebellum caused immediate relaxation of such tonus so long as the current was applied."

In 1902, Fröhlich and Sherrington¹⁷ attempted to trace the origin of the impulses which give rise to and which abolish the tonic state of the musculature in the decerebrate animal. They found that electrical excitation of the direct cerebellar tract in the spinal cord increased the rigidity of the homonymous limb, and that there existed a pathway in the ventrolateral column of the spinal cord, excitation of which resulted in the inhibition of rigidity in certain muscles. They concluded that "the path of inhibition in the ventrolateral white column may be connected with the afferent nerves from the crossed hind limb." Later, Sherrington¹⁸ found that one of the prime conditions necessary for the production of the tonic state in the decerebrate animal consisted in afferent impulses arising in the muscles themselves. "The de-afferented preparation," he writes, "exhibited no trace of decerebrate rigidity, the 'normal' preparation always did." Still later the same investigator¹⁹ proved that the decerebrate attitude was identical with the reflex act of standing; "That this is so may be demonstrated by setting the decerebrate animal on its feet; it is then seen that the preparation stands."

The experiments of Graham Brown²⁰ proved beyond a doubt that the center responsible for the tonic state of the decerebrate animal is the nucleus ruber of the midbrain. Section of the midbrain in front of the anterior colliculi resulted in extensor rigidity. Section below the posterior colliculi abolished the rigidity. Stimulation of various points in the cross section of the midbrain resulted in the assumption of definite postures of the body and limbs of the animal. These postures, moreover, continued to be maintained after the cessation of the

16. Löwenthal, M., and Horsley, Victor: On the Relation Between the Cerebellar and Other Centres (namely, Cerebral and Spinal), with Especial Reference to the Action of Antagonistic Muscles, *Proc. Roy. Soc.* **61**:20, 1897.

17. Fröhlich, A., and Sherrington, C. S.: Path of Impulses for Inhibition Under Decerebrate Rigidity, *J. Physiol.* **28**:14, 1902.

18. Sherrington, C. S.: On Plastic Tonus and the Proprioceptive Reflexes, *Quart. J. Exper. Physiol.* **2**:109, 1909.

19. Sherrington, C. S.: Postural Activity of Muscle and Nerve, *Brain* **38**:191, 1915.

20. Brown, T. Graham: On Postural and Nonpostural Activities of the Midbrain, *Proc. Roy. Soc.* **87**:145, 1913.

action of the stimulus. "The area includes that of the nucleus ruber and of the posterior longitudinal bundle. . . . Stimulation within this area . . . is accompanied by the assumption of a definite posture on the part of the animal," and again ²¹: "The main effect of stimulation of one red nucleus is a movement of flexion of the arm of the same side and a movement of extension in the opposite arm . . . those movements having attained their maximum, tending to be maintained long after cessation of the evocative stimulus."

The center for tonus having been thus located, the important question remained: What are the cerebral pathways which, in the normal state, keep the tonic function of the red nucleus in abeyance? The question was answered by Weed,²² who traced that pathway through the internal capsule, the crus cerebri, the middle cerebellar peduncle and the vermis of the cerebellum. That system is the frontopontocerebellorubral pathway. Electrical stimulation of this pathway resulted in an abatement of the decerebrate rigidity.

The conclusions of Weed were fully corroborated by later investigations. Thus, for example, Cobb and others,²³ give as one of the results of their investigations the following "Stimulation of the superior cerebellar arm in decerebrate cats causes ipsilateral inhibition of their extensor rigidity."

The state of tonus, then, depends on two sets of opposite impulses. One, the proprioceptive, arising in the muscles, passes along the spino-cerebellar tracts, and by way of the cerebellum, arrives at the red nucleus. The interruption of this pathway does away with the tonic activity of the red nucleus. The other set of impulses is of a kind which, in the presence of afferent stimuli, maintains the tonic activity of the red nucleus in abeyance. It takes its origin in the cerebral cortex and, by way of the cerebropontine system of fibers, passes into the cerebellum and so arrives at the red nucleus of the midbrain.

The cerebellum thus becomes the meeting place of two sets of impulses of opposite function. The uncertain results on the tonic function brought about by gross injuries to the cerebellum are easily accounted for by the fact that the fibers of the two pathways are, within that organ, very much intermingled, though more so in some situations than in others. This may be judged in part from the figure of the author's dissection (Fig. 1).

21. Brown, T.: On the Effect of Artificial Stimulation of the Red Nucleus in the Anthropoid Ape, *J. Physiol.* **49**:185, 1915.

22. Weed, L. H.: Observations on Decerebrate Rigidity, *J. Physiol.* **48**:205, 1914.

23. Cobb, S., Bailey, A. A., and Holtz, P. R.: Genesis and Inhibition of Extensor Rigidity, *Am. J. Physiol.* **44**:239, 1917.

A fact of great importance in connection with the state of rigidity is that the posture of the animal as a whole is determined by the position of certain of its parts. Thus Magnus and de Kleijn²⁴ showed that the position of the body of the decerebrate animal is changed by the passive rotation of its head. The removal of the

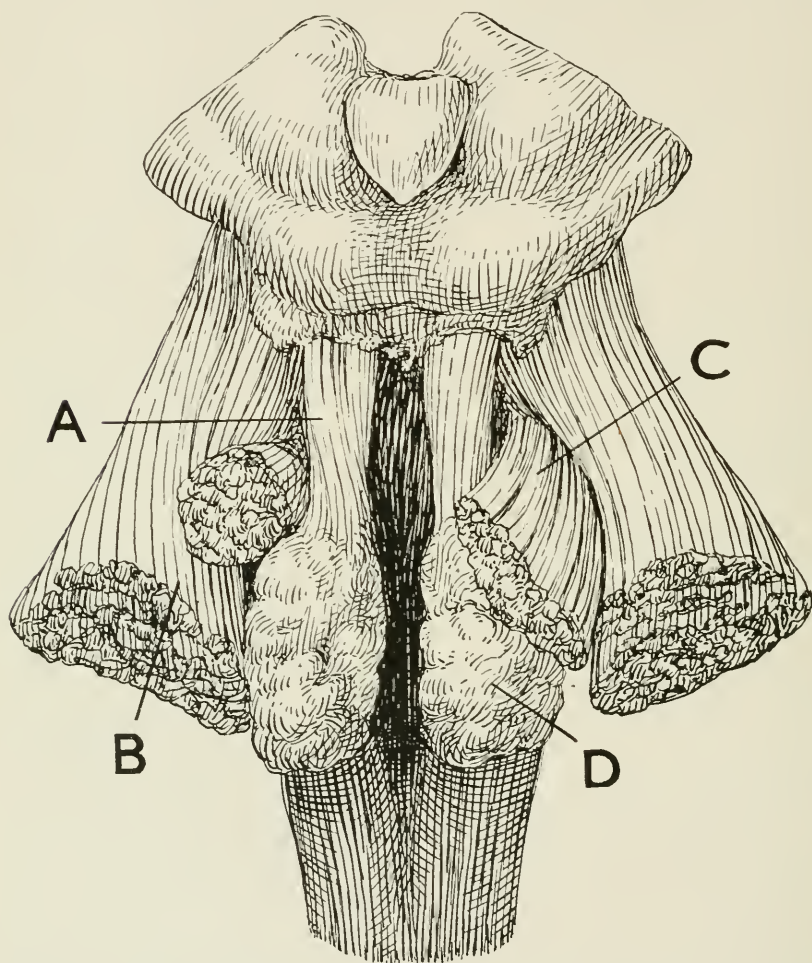


Fig. 1.—A dissection showing the relations of the peduncles to each other. *A*, superior peduncle; *B*, middle peduncle; *C*, inferior peduncle; *D*, dentate nucleus.

so-called neopallium alone with subsequent extirpation of one labyrinth results in a definite posture of decerebrate rigidity. Barenne and

24. Kleijn, A. de, and Magnus, R.: Beiträge zum Problem der Körperstellung, *Pflüger's Arch.* **180**:291, 1920.

Magnus²⁵ showed that when the head of such an animal is passively rotated, its body and limbs assume a different posture. Jonkhoff²⁶ describes a case of status epilepticus which illustrates the dependence of the posture of some of the muscles on that of others. The patient was unconscious and rigid. When the head, which was turned to the right, was passively rotated to the left, the extension of the right arm disappeared. These are examples of the manner in which the afferent impulses arriving from the muscles play on the center of tonus and, by accentuating the activity of certain of its parts, bring about a movement which results in a change of posture.

The vestibular mechanism, which plays a very important rôle in the maintenance of certain postures of the body, does not concern us in this connection.

The foregoing physiological facts have been turned to account by Wilson,²⁷ who has shown that a number of diseases in man associated with tonic rigidity or tonic spasms have been found at necropsy to be cases of practical decerebration, complete or partial. He concludes that: "Any lesion dissociating the cortex from the mesencephalocerebellar level and at the same time leaving the latter functionally intact is calculated to cause the rigidity to develop." In order to accentuate the importance of the inhibitory action of the cerebrum on the red nucleus, this investigator then adds that: "there is no question that the phenomena of decerebrate rigidity are release phenomena."

We may thus conclude that the inhibitory pathway in question comprises the entire cerebropontine radiation.

In order to obviate possible controversy, it is perhaps as well at the outset to throw one question out of court, namely, whether the action of a certain nerve impulse in extinguishing that of another is in the nature of an active inhibition or whether it is only apparently so. "A natural surmise," says Sherrington,²⁸ "seems that it may be in the nature of interference, somewhat as waves of sound or light by interference mutually extinguish each other." Bayliss²⁹ indeed explains this phenomenon on the physical basis of the extinction of a wave of force when two superimposed waves are so timed as to alternate in half phases; in other words, when the hollows of one are filled in by

25. Barrenne, J. G. Dusser de, and Magnus, R.: Beiträge zum Problem der Körperstellung, *Pflüger's Archiv*. **180**:75, 1920.

26. Jonkhoff, D. J.: The Prognostic Importance of the Magnus de Kleijn Reflexes in Man, *Nederl. Tijdschr. Geneesk.* **64**:307, 1920; abstracted in *J. Nerv. & Ment. Dis.* **55**:519, 1922.

27. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, *Brain* **43**:220, 1920.

28. Sherrington, C. S.: Reflex Inhibition as a Factor in the Coordination of Movements and Postures, *Quart. J. Exper. Physiol.* **6**:251, 1913.

29. Bayliss, W. M.: *Principles of General Physiology*, 1915, p. 420.

the protuberances of the other. Keith Lucas³⁰ has shown such an interference of one nerve impulse with that of another to be equivalent to an impulse passing through a region of increasing decrement. In the course of a study of the simplest reflex arc, Veszi³¹ was able to demonstrate the fact that a muscular contraction induced by the excitation of one posterior nerve root may be entirely inhibited by the excitation of another posterior root. Tilney³² offers the simple explanation of the complete neutralization of one force by another in a parallelogram in which forces of the same magnitude work in opposite directions.

The important fact bearing on the subject with which we are concerned is that the moderation of the activity of the red nucleus, in the normal state of the adult, depends on certain cortical activities propagated to the midbrain; and that when these activities are suspended, the nucleus of the midbrain, no longer impeded in the exercise of its function, brings about a state of general muscular rigidity. The exact nature of the cortical activities which impede the function of tonus of the red nucleus is quite immaterial from the standpoint of the present study.

Although the midbrain occupies a position of preëminence in the function of tonus, its monopoly over this function is not complete. The musculature of the dog or cat whose spinal cord has been transected,¹⁸ recovers after a time a certain amount of its former tonicity. Pike³³ attributes this partial recovery to structures in the cord which remain dormant while the more highly evolved mechanisms can perform the same function in a more efficient manner, but which become active when these are removed. It is undoubtedly true that during the course of evolution of the nervous system, the transference of certain functions from lower to higher levels has gone far in advance of the transference of the structures with which those functions are associated. For while we know that the course of organic evolution is characterized not only by aggregation but by a simultaneous segregation of structures and functions as well, the latter process is practically never complete.

THE BIOLOGIC SIGNIFICANCE OF POSTURE. THE NORMAL PERIODS OF SUSPENDED CEREBRAL ACTIVITY

The special importance of posture arises from the fact that animals are made up of jointed segments. With respect to the decerebrate

30. Lucas, Keith: *The Conduction of the Nervous Impulse*, 1917, p. 93.

31. Veszi, J.: *Der einfachste Reflexbogen im Rückenmark*, *Zeitschr. f. allgm. Physiol.* **11**:168, 1910.

32. Tilney, Frederick: A personal communication.

33. Pike, F. H.: *Studies in the Physiology of the Nervous System*, *Am. J. Physiol.* **24**:124, 1909.

animal it is undoubtedly true that¹⁹ "the distribution of this reflex tonicity embraces just those muscles whose contraction tends in the erect position of the animal to counteract the effect of gravity." With respect to the intact animal, however, the function of tonus has another equally important use. In order to enable any distal segment of the body or the limbs to execute a measured movement, the central segment to which it is jointed, must be fixed. Thus in order that the forearm may move for a definite distance in a definite direction, the arm must be fixed at the shoulder. Fixation of the shoulder, complete or partial, according to the necessity of the occasion, is accomplished by a tonic contraction of the muscles which pass over the joint on opposite sides. And it is this fixation which constitutes the special posture of the upper limb in certain movements of the forearm.

In the ordinary activities of the animal, occasions must be very numerous when joints are partly or wholly immobilized for varying durations of time by the contraction of the overlying musculature, in order to afford a steady base for the movement of relatively distal segments of the body and limbs. We have seen that the special nerve center which presides over the production of this fixation of the joints—over the maintenance of posture—is the red nucleus. We have further seen that the activity of that nucleus, in the presence of certain stimuli, is dependent on the extinction of certain impulses arriving from the cerebral mantle. Every possible movement of the animal, implying as such movement does the temporary tonic contraction of certain groups of muscles, must also imply a simultaneous temporary extinction of certain cerebral activities. When considering the epileptic paroxysm as it occurs in the severer form in the epileptic, and in the milder form in all normal persons, we shall have an opportunity for a more concrete application of the foregoing facts.

MOVEMENT (CLONIC MUSCULAR CONTRACTIONS) AS A MODE OF REDISTRIBUTION OF TONUS

In order to explain the phenomenon of movement, as distinguished from that of posture, certain investigators have found it necessary to postulate the existence of special anatomic structures, outside of those devoted to the function of tonus and the inhibition of tonus.

"While a state of momentary decerebration," writes Sargent,³⁴ "may be held to explain the tonic stage of the epileptic fit it is not easy to account for the clonic movements. Kinnier Wilson . . . having characterized the tonic fits he observed in the cases which he was describing as 'attacks of decerebration,' remarks that the absence of any clonic movement is of much significance. He takes it to indicate that clonic movements have different origins and are the expression of

34. Sargent, P. W. G.: Some Observations on Epilepsy, *Brain* **44**:312, 1921.

the activity of different motor mechanisms, the one being characterized by static, the other by phasic activity."

J. Ramsey Hunt³⁵ postulates "two distinct components, each represented throughout the entire efferent nervous system by separate neural mechanisms which are physiologically and anatomically distinct. One is the movement proper, which is subserved by the *kinetic system* (motion system). The other represents that more passive form of contractility which we recognize in tonus, posture and equilibrium, and is subserved by the *static system* (posture system)."

In the light of the known anatomic and physiologic facts, the assumption of special systems devoted to *movement as a distinct physiologic entity*, appears to be quite gratuitous, as may be gathered from the following considerations:

Since the common final motor pathway has its origin in the ventral gray columns of the spinal cord and brain stem nuclei, it must be plain that the probable action of the red nucleus, however indirectly, is on the motor cells of the cord. Wilson²⁷ indeed concludes that "we cannot believe that in the red nucleus we have other than a re-presentation of the ventral cord cell groups," and that "stimulation of a point in the red nucleus . . . may be supposed to innervate a physiologic group of ventral cells."

It is a fact that injury to the cerebrospinal tract in any part of its course results in a state of increased tonus of the musculature below the level of the lesion.

If the tonic action of the musculature is to be ascribed to the influence of the red nucleus on the motor cells of the spinal cord, then the increased tonus resulting from pyramidal lesions must be attributed to removal of an inhibitory influence which that tract normally exerts on the ventral motor cells. In this respect, therefore, the cerebrospinal tract must exercise an influence on a lower level similar to that which the cerebropontine tract exerts on a higher level. Such an assumption, it will be presently seen, is not in the least incompatible with the fact that stimulation of the area of origin of the cerebrospinal pathway—the precentral—results in movement.

The influence of the pyramidal fibers on the ventral cells of the cord is most probably by means of an intermediate neuron. Each of the pyramidal fibers is thus unable to transmit an impulse to a number of ventral cells. We know, moreover, that the groups of cells thus innervated by the pyramidal fibers correspond to definite groups of muscles. The simplest volitional movement, say that of flexion at the elbow, involves the synergic contraction of a number of flexor muscles with a simultaneous inhibition of a number of extensors. The details of

35. Hunt, J. R.: Dyssynergia Cerebellaris Myoclonica, Brain **44**:490, 1921.

the mechanism of reciprocal innervation of antagonistic muscles which Sherrington³⁶ called "co-operative co-ordination" cannot but be found in a patterned arrangement of groups of motor cells in the spinal cord.

The particular force which brings about muscular contraction is, in our ignorance of its ultimate nature, known as muscle tonus. When the amount of tonus of one set of muscles is exactly counterbalanced by an equivalent amount of tonus of an antagonistic set, the limb remains at a standstill, in a definite posture. Any difference in the amount of tonus of the two respective sets of muscles—any difference of potential—manifests itself in movement; and the movement continues until a balance between the forces is again established. At that moment the limb is once more in a definite posture.

We are thus enabled to understand why electrical excitation of the precentral area is productive of movement. A stimulus passing along the pyramidal fibers to the cells which innervate, say, a group of extensors, must cause an abatement of the extensor tonus. The consequent predominance of flexor tonus will then result in a movement of flexion. Once the contraction of flexors has begun, it will proceed with an increment of force for the following reason:

The tonic action of the nucleus of the midbrain depends on certain afferent impulses. These impulses have been shown by Sherrington¹⁸ to originate in the muscles themselves. The condition for their origin is the approximation of the points of insertion of the muscles. The beginning contraction of a muscle, therefore, by propelling stimuli to the midbrain, increases the tonic activity of the red nucleus and receives from it in turn increments of force.

Again, we know that stimulation of various points in the tonic mechanism results in the assumption of definite postures. Stimulation of different degrees of intensity and of different points in that mechanism must result, therefore, in *changes* of posture; and movement is nothing but a succession of such changes. These, we have seen, may result from alterations in the activity of the proprioceptive mechanism, as in the Magnus and de Kleijn reflexes. The rhythmic movements of progression of Weed's³⁷ decerebrate kittens exemplify the manner in which a succession of proprioceptive stimuli is capable of producing a succession of postures. And the same is true respecting the coordinate movements obtained by Graham Brown³⁸ in spinal and decerebrate preparations. Other factors are equally potent in producing changes of

36. Sherrington, C. S.: On Reciprocal Innervation of Antagonistic Muscles, *Proc. Roy. Soc.* **60**:414, 1897.

37. Weed, L. H.: The Reactions of Kittens After Decerebration, *Am. J. Physiol.* **43**:131, 1917.

38. Brown, T. G.: Studies in the Physiology of the Nervous System, *Quart. J. Exper. Physiol.* **6**:25, 1913.

posture, as may be gathered from the following:²⁸ "Take the case where a reflex (decerebrate) preparation is exhibiting the steady postural reflex of standing, and then draws up one foot on that foot being pinched. . . . In the transition from one reflex act to another, a muscle's activity is inhibited if it would offer obstruction to the new reflex."

Our conception of movement, then, is simply one of redistribution of tonus—the leap from one posture to another. Such a conception corresponds with the effect of the process on the environment. This effect is *work done*, which John Stuart Mill³⁹ defined as *a change in the position of objects*.

Such a conception of movement, too, enables us to understand how localized clonic contractions of muscles may be caused by localized injuries of the motor area of the cortex. When certain points in the tonic mechanism of the midbrain or of the spinal cord are released from the impeding action of certain portions of the cerebropontine or the cerebrospinal systems, their functional activity remains determined by afferent impulses only. The periodic ebb and flow of such afferent impulses must therefore result in periodic convulsions of the parts of the body whose musculature is presided over by corresponding patterns in the tonic mechanism.

THE RELATION OF MUSCULAR ACTIVITY TO THE CONSCIOUS STATE

Before proceeding with facts regarding the occurrence of the several forms of the epileptoid paroxysm in normal persons, the relation in which muscular contraction stands to the conscious state must be elucidated.

The terms consciousness and unconsciousness, as employed in the present study, are meant to convey but a general meaning. The former, as it is applied to the wide-awake state of the average normal person; the latter, as it might be applied to profoundly comatose conditions; and the innumerable degrees of transition between the two states, as these might exist in a person who passes from the normal waking state into one of profound coma.

That merely coordinated movement is not in itself a measure of the degree or scope of the conscious state, is evidenced by the movements executed in states of automatism, in delirium, as well as by the animal with an ablated cortex, or by the spinal frog. Even when such movements subserve both immediate and distant benefits, they are not necessarily a sign of the fully conscious state. Thus, a pedestrian, on

39. Mill, John Stuart: *On the Requisites of Production*. Principles of Political Economy, 1891.

his way to transact some business previously determined on may, while walking, be in that semiconscious state which is known as "absent-mindedness." Yet his act of walking, automatic as it is, may subserve both immediate and distant benefits.

All such examples of movement, no matter how simple or how complex are, however, marked by the common characteristic that at the time of their execution they are beyond the control of the individual. From the viewpoint of movement, therefore, it is the ability to control muscular contraction which is the measure of the degree of the conscious state at any one time.

Much less obvious than the preceding is the proposition that even though a muscular contraction be initiated voluntarily, the contraction itself implies a certain reduction in the conscious state which is in proportion to the intensity of the muscular activity. Without an appreciation of the facts involved in this proposition it is not possible to understand the connection between the reduced state of consciousness and the muscular phenomena either in the normal epileptoid or in the epileptic paroxysms. It is, therefore, important to deal with them in this place.

The fact that even very strong stimuli may be prevented from reaching the sphere of consciousness by voluntarily initiated muscular contractions, bears testimony to the truth of the foregoing proposition. Darwin ⁴⁰ called attention to the incompatibility of the conscious state and that of muscular activity as follows: "A man cannot think deeply and exert his utmost muscular force. . . . Sailors who are going to be flogged sometimes take a piece of lead into their mouths in order to bite it with their utmost force and thus to bear the pain. Parturient women prepare to exert their muscles to the utmost in order to relieve their sufferings."

Whoever has suffered from an intense toothache will testify to the fact that the pain is much more severe when resting quietly in bed than when "pacing the floor." Persons afflicted with worry "pace the floor" and are generally "on the go." Such muscular exercise, by reducing in part the conscious state, offers them a degree of relief.

A probable explanation of such a relation of muscular contractions to varying states of consciousness may be gathered from the consideration of the production and distribution of tonus:

We have seen that one of the conditions for setting in action the tonic mechanism of the midbrain is a suspension of the function of the cerebropontine system. That system is the largest cerebral efferent fiber system. It descends from a number of points throughout the

40. Darwin, Charles: *The Expression of the Emotions in Man and Animals*, 1873, p. 72.

extent of the cerebral cortex, with the possible exception of the lowest portion of the temporal and occipital regions. Its extent may be judged in part from Figure 2 of the author's dissection. The abandonment of function by so widely distributed a system of neurons must result in profound dissociations of the neural mechanisms with which it is connected in the cerebral cortex; and such dissociation is equivalent to a reduction in the conscious state.

The truth of the foregoing considerations may be tested in several ways. If the reduction of the conscious state is a necessary condition for the production of muscular contractions, then any degree of narrowing the field of consciousness should result in some degree of muscular activity, and different degrees of contractions of the field of consciousness should result in corresponding degrees of muscular activity.

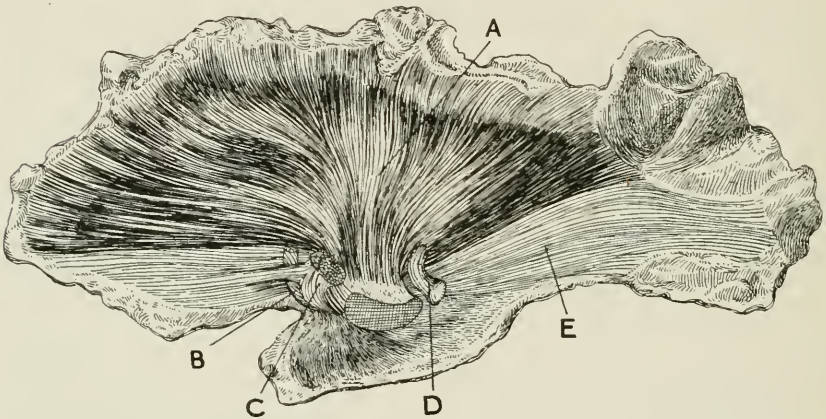


Fig. 2.—Mesial view of the cerebral structures exposed on removal of the thalamus and the thalamic fiber system. *A*, pallioptine and pyramidal systems; *B*, anterior commissure; *C*, pallidal fiber system *X*; *D*, internal geniculate body and auditory tract; *E*, an *X* fiber system converging on the globus pallidus.

The field, or scope of consciousness may be narrowed by the act of concentration of attention. That the latter act really implies a contraction of the field of consciousness may be easily gathered from the fact that the person whose attention is "absorbed" is to a great extent unconscious of his surroundings and that he may indeed be insensitive to a number of even very strong stimuli. It is a familiar fact that when such a person, by dint of repeated requests, is finally aroused to reply, he manifests a total ignorance of the previous attempts to arouse him.

The act of concentration of attention is accompanied by certain muscular activities. Lindley's ⁴¹ study of 622 pupils in the schoolroom

41. Lindley, E. H.: A Preliminary Study of Some of the Motor Phenomena of Mental Effort, *Am. J. Psychol.* 7:491, 1895.

led him to the conclusion that varying degrees of mental concentration manifested themselves in certain sustained postures and automatic movements. Stein⁴² describes clonic movements which she observed when attention wandered, that is to say, when it shifted from point to point. The following two experiments, performed by the author a number of times, may be repeated by anybody with little trouble and much profit.

The first consists in concentrating the attention on a single point or on the succession of a single sound, such as the falling of water drop by drop. It will then be found that as long as attention is sustained, a single posture of the body and the limbs is sustained as well; and the posture is involuntarily changed immediately attention begins to shift. The reverse is likewise true. Any attempt to change the fixed posture results in the shifting of the attention.

The second experiment consists in the solution of a problem involving sets of facts each of which requires separate careful consideration. It will be found that simultaneously with the shifting of the thought from one set of facts to another, there is an involuntary change of posture.

Féré⁴³ found that the concentration of a person's attention by sharp and penetrating odors increased his capacity for muscular exertion. The so-called psychogalvanic reflex has its origin, in part at least, in the muscular contractions which ensue when the scope of consciousness is reduced within certain limits by definite sensations, or emotions. By an improvement of Hamlet's⁴⁴ method, Peterson and Jung⁴⁵ obtained an electrical response when certain words which compelled the subject's special attention were read to him. The findings of these investigators, as well as the similar findings of Féré,⁴⁶ of Veraguth,⁴⁷ and others, were at first attributed to other causes than muscular activity. Sidis and Nelson,⁴⁸ however, in their experiments

42. Stein, G.: Cultivated Motor Automatism, *Psychol. Rev.* **5**:295, 1898.

43. Féré, C.: Les variations de l'excitabilité dans la fatigue, *L'année psychol.* **7**:69, 1900.

44. Hamlet. " . . . The play's the thing
Wherein I'll catch the conscience of the King.
.
One scene of it comes near the circumstance
Which I have told thee of my father's death.
I prithee, when thou seest that act a-foot,
Even with the very comment of thy soul
Observe my uncle . . . "

45. Peterson, F., and Jung, C. G.: Psychophysical Investigations with the Galvanometer, *Brain* **30**:153, 1907.

46. Féré, C.: Note sur des modifications de la résistance électrique sous l'influence des excitations sensorielles et des émotions, *Compt. rend. Soc. de Biol.* **5**:217, 1888.

47. Veraguth, O.: Abstracted in *Arch. d. Psychol.* **6**:162, 1916.

48. Sidis, Boris, and Nelson, L.: The Nature and Causation of the Galvanic Phenomenon, *Psychol. Rev.* **17**:98, 1910.

on animals came to the conclusion that the electrical current was produced by muscular contraction. In summing up the results obtained by different investigators in this line of research, Prideaux ⁴⁹ concludes that the so-called psychogalvanic reflex which is concomitant with the experience of certain emotions is, in part at least, due to muscular contraction. It is, therefore, in part, really what is now known as a muscle action current.

The relation between muscular contractions and the conscious state, therefore, may be likened to the two arms of a lever; the ascent of one arm involves the descent of the other. Each muscular contraction, implying as it does a release of muscle-tonus, implies at the same time a suspension of a certain amount of cerebral activity—a certain reduction in the conscious state.

III. THE NORMAL EPILEPTOID REACTION

The phenomena which constitute the normal epileptoid paroxysms, like those associated with true epilepsy, exhibit a wide variation in the intensity and duration of each attack as a whole, as well as in the separate manifestations. Thus, for example, that stage of the epileptic attack during which the disintegration of the patient's consciousness marks its progress on the way to extinction, may be of such long duration, that the dominant symptom of the attack is one of elaborate reactions to equally elaborate hallucinations. On the other hand, the transition from the wide-awake state to that of utter unconsciousness may be sufficiently rapid to escape the notice of the keenest observer. And the same is true of the other manifestations of the attack. Notwithstanding, however, the wide variation in the degree of intensity and duration of the separate manifestations of the epileptic paroxysm, the sequence in which they follow each other in the more pronounced cases is, on the whole, uniform. This sequence is as follows: (1) A preliminary stage, which consists of a reduction in the conscious state. (2) A state of muscular contraction, such that the tonus is equally distributed to antagonistic muscles, except for some predominance of certain extensors, pronators, and adductors. This equal distribution of tonus to antagonistic muscles results in immobilization of the joints and a consequent state of rigidity. When the rigidity is general and its progress rapid, its onset is marked by an expiratory sound, the epileptic cry. (3) A redistribution of the muscle-tonus, resulting in clonic movements. During this stage involuntary evacuation of the bladder or the rectum may take place. (4) A period of recovery, marked by exhaustion and a gradual integration of the previously disintegrated state of consciousness.

49. Prideaux, E.: The Psychogalvanic Reflex, *Brain* **43**:50, 1920.

These phenomena, in the same sequence in which they follow each other in the epileptic attack, occur also in all normal individuals under two sets of conditions: (1) on exposure to the wide range of circumstances which necessitate sudden movement, or the sudden concentration of attention; (2) in connection with the performance of certain functions. When considering the causes of the periodic recurrence of the epileptic paroxysm, it will be shown that the normal epileptoid paroxysm, no matter what its determining factors may be, is subject to the same law of periodicity.

THE EPILEPTOID REACTION IN RESPONSE TO EXTERNAL STIMULI

The general condition for the occurrence of the epileptoid reaction to be described under this heading is exposure to a stimulus so sudden as to take the person or the animal unawares.

The following case, although rather unusual, illustrates the severe form of the normal epileptoid paroxysm. The milder and the far more common forms which occur in the daily life of every person, will be pointed out in the consideration of the separate manifestations of the fit.

A young school teacher, James B., met with an accident on a farm in Woodstock, Ulster County, N. Y., in the fall of 1922. While picking apples, the branch on which he stood gave way. He fell from a height of some twelve feet and fractured three ribs. The young man was of light weight and agile, and the ground on which he fell was soft; it seemed therefore singular that he should have sustained so much injury. As the accident took place while the present study was in progress, the writer was especially interested, and the injured man as well as the members of his family, who were with him at the time of the accident, were questioned regarding the details of the occurrence. The following history was elicited:

The patient, healthy and intelligent, 23 years of age, stated that while picking apples in a tree, he suddenly heard the branch on which he stood crack. He next became aware of standing with a cup in his hand from which he was drinking water. It then occurred to him that something unusual must have happened, for he was surrounded by the members of his family who appeared to be solicitous about his condition. He remembered that he had been picking apples in the tree and that he had heard the branch break, and realized that he must have been unconscious for a time, for the spot where he stood with the cup in his hand was at a considerable distance from the fateful apple tree. He had no direct knowledge of the fall. Any such previous attack of unconsciousness was absolutely denied, and the nature of his occupation lends credence to this denial.

The members of his family, who were with him at the time of the accident, stated that James was picking apples in the tree when they heard him cry out and saw him fall to the ground. They ran to him and helped him rise. Although he appeared rather dazed and did not answer their questions, yet when led along towards the house, he appeared to walk in a fairly normal manner. After walking some distance, he asked for water, and when it was brought to him he drank it. A little later he complained of pain at the site of the injury.

The separate phenomena involved in this case will be considered in the succeeding sections.

1. *The Stage of Disintegration of the Conscious State. The Aura.*—The immediate effect of exposure to a sudden painful or emotional stimulus is a rapid contraction of the field of consciousness to within the limits occupied by the stimulus. It is the process known as “centering the attention.” The extent and rapidity with which the field of consciousness is thus narrowed is dependent on the strength of the stimulus and the suddenness of its application on the one hand, and on the state of unpreparedness of the person on the other. In the experiments on the effect of suddenly applied stimuli made in connection with the present study the second stage, that of general rigidity, came on apparently without being ushered in by any reduction in the conscious state. In these experiments, however, the stimulus, though unexpectedly and rapidly applied, was necessarily very weak and the spasm of general rigidity, though very distinct, was at the same time exceedingly brief. It is to be expected, therefore, that the stage of unconsciousness in the case of exposure to the weaker kinds of emotional or painful stimuli, would largely escape observation. Cases of moderate fright, or of other emotional shocks, such as the hearing of a joyful or a distressing piece of news are, however, on record in which the initial reduction of the conscious state is very considerable. The accident related in the preceding section is a fair example of a class of cases in which the reduction of the conscious state is pronounced and the progress of the reduction rapid. When, however, such a reduction of consciousness is very gradual, it is a rule—and that rule holds good to a very large extent in epilepsy as well⁵⁰—that it does not sink to the point of complete extinction. In such cases of gradual disintegration of consciousness, hallucinations and automatism make their appearance.

Auditory hallucinations occurring in states of “emotion” or “excitement” are mentioned by Schwartze,⁵¹ Politzer,⁵² and others. The former author describes these “physiological subjective auditory sensations” as occurring most frequently in the form of ringing sounds of different tone and loudness, appearing with great suddenness and vanishing gradually; more rarely these sounds consist of a number of tones which are repeated over and over again. One of the writer’s

50. Gowers, Sir William R.: *The Borderland of Epilepsy*.

51. Schwartze, H.: *Ueber subjective Gehörsempfindungen*. Berlin. klin. Wehnschr., No. 12, p. 124, 1866.

52. Politzer, Adam: *Ueber subjective Gehörsempfindungen*. Wien. med. Wehnschr. No. 94, 1693, 1865.

patients, however, described his auditory sensations as resembling the noise of a bursting steam boiler with a subsequent hissing sound.

A number of the cases of sudden subjective blindness which have been conveniently stacked away under the heading of "hysterical amauroses" may be discovered, on a closer examination of the circumstances under which they occurred, to consist of hallucinatory experiences associated with the epileptoid reaction. The following case from Willbrand and Saenger⁵³ is an example:

A seamstress, 20 years of age, had broken the needle while sewing on a machine, and a fragment flew off in the direction of her left eye. She immediately experienced a pain and lost vision completely in that eye. On examination the eye was found to be uninjured. Vision returned within three days.

Secular as well as religious literature is full of instances of visual hallucinations occurring in connection with states of surprise, anxiety and fear. The following from Job (iv, 14) is an example:

"Fear came upon me and trembling, which made all my bones shake, Then a spirit passed before my face. . . . It stood still but I could not discern the form thereof: an image was before my eyes. . . ."

Cutaneous hallucinations—paresthesia—are exceedingly common even in the very mild cases in which the duration of the entire paroxysm is only a second or two. These are commonly described by the person as a sensation of numbness in the extremities or as that of cold running down the back. The "shiver running down the back" may be a muscular reaction to a cutaneous hallucination of central origin, or its origin may be local and induced by certain vasomotor changes associated with the attack. When the entire paroxysm is brief, these sensations outlast the tonic and clonic stages and persist during the stage of recovery.

Hallucinations of deep sensation may be easily inferred from such statements as: "I could not find my limbs," or "I lost all feeling in my limbs," or "I was afraid my legs would give way under me." The lower limbs do indeed sometimes give way in the first stages of the paroxysm of severe fright, extreme annoyance, or intense joy. Such muscular collapse may be considered as a reaction to an hallucination of the deep or muscle sense. In the instance of the accident to the school-teacher described, the fall may have been due either to such a reaction, to an hallucination of muscle sense, or to the second stage of the paroxysm, that of general rigidity. It may be remarked in passing, that the dropping of the upper limbs to the sides of the body which is the ordinary gesture of real or feigned surprise in common social intercourse, may have its origin in the normal epileptoid paroxysm—

53. Willbrand, H., and Saenger, A.: *Neurologie des Auges*. 3:1006, part 2, 1906.

a gesture whose familiar meaning is meant to be readily recognized by the observer.

Vestibular hallucination—subjective vertigo—is a very frequent symptom in the first stage of the epileptic attack. It is sometimes the only manifestation of the disease for certain periods of time, as may be seen from Case 7 (p. 731). The sensation of vertigo or dizziness is perhaps the most frequent symptom of that reduction or disintegration of cerebral function, which precedes the muscular phenomena of the normal epileptoid paroxysm consequent upon exposure to sudden painful or emotional stimuli. Such popular expressions as “dizzy with joy,” “dizzy with fright,” or “dizzy with surprise,” and a hundred other variations of the same expressions testify to the frequency of vestibular hallucinations in the first stage of the paroxysm associated with these normal conditions.

Epigastric hallucinations are exceedingly common. In the experiments to be mentioned later, the facial expression was frequently one of disgust, which sometimes persisted for a number of seconds. The reason for it given by the subject was that the procedure had made him or her “a little sick at the stomach.” At other times the facial expression was one of uneasiness and the reason offered for this was a “peculiar sensation” of an indescribable nature within the epigastrium.

In cases of epilepsy in which this stage is of sufficient duration, automatism is frequent. An ordinary example of it may be seen in the following observation of Case 12 (p. 733) from the Vanderbilt Clinic Series:

When requested to take off his shoes for the purpose of testing the Babinski reflex, he stared at the writer in an astonished manner for a moment, smiled foolishly and began to rub the back of his head in a persistent manner. He then turned away his face, smiling all the time, and making chewing movements. A minute later he rose from his seat and proceeded to walk. When half way across the room he turned around. His face was now expressionless. When led by the examiner to his seat, he offered no resistance. He then became rigid for a moment and a barely perceptible tremor ran over him. Gradually, in the course of two or three minutes, consciousness began to return and in another minute or so he was sufficiently recovered to respond to the request to remove his shoes.

Cases of epilepsy exhibiting far more complicated movements have been described by a number of investigators. One or two such cases from the writer's series will be given later.

In the case of the normal paroxysmal reaction to sudden emotional or painful stimuli, automatism may be observed with surprising frequency during the first stage, if the latter is sufficiently prolonged. The purposeless movements of the surprised or frightened person, which consist in rubbing the chin, in scratching the neck, in picking up objects and replacing them, in chewing movements, in walking

movements, in irrelevant smiling or giggling, and in the hundred other such unmotivated yet perfectly coordinated movements, are familiar to everybody and need no further description.

The analgesia which occurs during this and the subsequent stages of the normal epileptoid reaction will be dealt with later. Suffice it to say at this point that such insensibility to pain has the implication of a contraction of the field of consciousness.

2. *The Stage of Tonic Rigidity.*—This is the most constant as well as the most striking manifestation. It may be observed in cases of exposure to the sudden action of almost any stimulus whether sensory or emotional, from the mildest to the most intense.

The experiments carried out in this connection were of the simplest kind. The subject stood with his back to the observer. He was asked to relax his muscles as much as he could. The back of the concha of the subject's ear was then sharply struck by the observer's finger nail. In an instant he assumed a state of general tonic rigidity which was more pronounced in women and children than in adult men, and much more so under circumstances to be detailed later. The back was straightened, the head raised; the thighs and legs were extended, the shoulders were elevated, the upper limbs were extended and pronated; the fingers were either extended on the contracted palm or half closed in a partial fist; the thumb was adducted against the palm.

It will be remembered that a characteristic feature of the tonic rigidity of the epileptic fit is the adduction of the thumb against the palm in such a manner that the fingers are flexed over it. Gowers⁵⁴ calls attention to this fact. Spratling¹⁰ mentions it as among the signs which differentiate the true epileptic fit from malingering. The position of the thumb in the condition of decerebrate rigidity described by Turner⁵⁵ was across the palm. In the previously mentioned case of tonic rigidity described by Stewart and Holmes, the flexed fingers covered the thumb. In one instance of the experiments in question the flexion of the fingers was complete, and the thumb of the hand observed was covered by the flexed fingers.

The sudden prick of a pin produced the same result, but the method is obviously disagreeable both to the subject and to the observer. The application of cold to the body, though remarkably adequate in evoking the epileptoid reaction in a number of persons, has been repeatedly tried and was finally discarded as difficult to apply with the needed degree of suddenness. The fact that a little time elapses before the subject experiences the low temperature of the tube (about half a

54. Gowers, Sir William R.: *Epilepsy and Other Chronic Convulsive Diseases*, 1901, p. 88.

55. Turner, C. Violet: *A Case of Prolonged Hyperpyrexia in a Child with a Midbrain Tumour*, *British J. Child. Dis.* **13**:261, 1916.

second), as well as the persistence of sensation after its removal, introduces confusing factors. No experiments were made with loud sounds or sudden flashes of bright light for the reason that the effects of these stimuli may be studied almost anywhere. Everybody who travels in public conveyances has noticed that the sound of a bursting tire impels a number of the passengers, particularly of the female sex, to leap for a moment into the characteristic attitude of decerebrate rigidity. Everybody has seen women drop objects which they hold in their hands on exposure to any one of the numerous stimuli comprised under the category of "surprise." Whoever will take the trouble to observe his own reactions at times when the highest functions of the nervous system are on the ebb, will find himself bounding into the decerebrate attitude on the slamming of a door, a clap of thunder, a flash of lightning, or a friendly slap on the shoulder from behind.

It goes without saying that the duration of these attacks is exceedingly brief—momentary. Yet brief as the attack may be, the sudden change of posture from that of relaxation to one of general rigidity is always strikingly pronounced.

The condition known as myotonia congenita is characterized by the tardy relaxation of normally contracting muscles. To persons who are subject to this affliction, the normal brief paroxysms of general tonic rigidity becomes a source of embarrassment and danger. The onset of the tonic attack is caused in such persons under the same conditions and with the same rapidity as in persons with a normal musculature, but the myotonic muscles, once contracted, are not relaxed with the same degree of despatch. The author has observed one such attack in a myotonic person, brought on by the application of a weak faradic current.⁵⁶ The rigidity which ensued lasted for two or three minutes and was singularly pronounced, the consistency of the muscles being more that of wood than of flesh. Thomsen⁵⁷ has described such patients as falling on exposure to relatively trivial emotional stimuli, and the cases described by Bell⁵⁸ are certainly of the same kind.

The most powerful of the emotional stimuli capable of inducing a state of rigidity in normal persons is by all odds that of fear. Such popular expressions as "scared stiff," "petrified with fear," and others of the kind, testify to the potency of fear in inducing a state of high and equal tonus in the antagonistic sets of muscles throughout the body. The state of sudden anger ranks in this respect perhaps only next to fear.

56. Rosett, Joshua: A Study of Thomsen's Disease, *Brain* **45**:1, 1922.

57. Thomsen, J.: Tonische Krämpfe in willkürlich beweglichen Muskeln, *Arch. f. Psychiat.* **6**:702, 1876.

58. Bell, Sir Charles: Quoted by A. McL. Hamilton in the *Med. Rec.* **29**: 85, 1886.

The hunting of deer at night by means of a jack-light is prohibited by law in a number of our states. The writer was informed by a colleague, who witnessed a deer exposed to the glare of a strong light at night, that the animal thus surprised stood stock still and could have been easily shot or taken in that condition. The writer himself has repeatedly seen rabbits by the roadside at night, in the glare of an automobile light, remain immobilized by the sudden action of the stimulus. Nor can it be said that animals who live in the sunlight are not trained to react in the presence of light. That their lack of training is with respect to the suddenness rather than to the nature of the stimulus applied, is borne out by the experiments on persons mentioned above. When the subject had been previously informed of what was to be done to him, no reaction, or only a very slight reaction took place.

The vast utility of the reaction of general rigidity on exposure to the sudden action of a stimulus will appear in a subsequent section.

The Paroxysmal Outcry: In the more pronounced instances of general rigidity induced in connection with the present study, the instant of onset was marked by an expiratory sound, "ah" or "oh"—the familiar sound of surprise. A similar outcry marks the onset of the tonic stage of the classical epileptic fit. In both cases it is caused by the sudden contraction of the muscles of the diaphragm, the abdomen and the thorax, and of those muscles which obliterate the opening of the larynx. The result is that a column of air is forced through the approximated vocal chords. The prolonged, unearthly cry of sudden intense fear is brought about by a similar mechanism. The ordinary "Ah!" or "Oh!" of pretended surprise in polite conversation almost certainly has its origin in the paroxysm of general rigidity ensuing in the momentary normal states of decerebration.

Anesthesia Associated with the Paroxysm: The inadequacy of stimuli to make themselves manifest as sensations in that state of reduced consciousness which is associated with the epileptoid reaction to "surprise" is very striking. Every physician in active practice can relate instances of patients who had received painful injuries during states of excitement and who were quite unconscious of any pain until some time later. The case of Livingston, who experienced no pain while his arm was bitten by a lion, is a classical example. Rivers⁵⁹ tells of the complete cessation of pain in the inflamed skin over his shins during excitement when the vessel in which he sailed was threatened with shipwreck. The writer himself had the following experience:

While suffering from an intense toothache in an isolated house in the mountains one early morning, he heard the cry of the cook downstairs that the

59. Rivers, W. H. R.: *Instinct and the Unconscious*, Cambridge University Press, 1922, p. 58.

oil stove was aflame. He ran down, put out the flame, then returned and proceeded to dress for breakfast. The idea of dressing for breakfast seeming strange, he recollected that some time earlier in the morning he had been annoyed by the thought that he would not be able to eat breakfast on account of the toothache. It then dawned on him that the intense pain had entirely disappeared. It was not before the expiration of fifteen minutes or more that the gradually returning pain attained its former intensity.

Similar experiences may be found on all sides and need not be dwelt on.

3. *Clonic Movements*.—In the experiments on the effect of the sudden application of a stimulus, mentioned above, the momentary state of general tonic rigidity was followed in about half the cases by clonic movements. They were observed in the hands and fingers and were of the choreo-athetotic type—a few movements of small amplitude and little force. In this respect, therefore, the experiments, in which the stimulus employed was of necessity very weak, are not conclusive. Far more definite was the occurrence of clonic movements following the state of rigidity which the writer has observed in the normal paroxysmal reactions in the ordinary experiences of every day life. A mere suggestion of the familiar circumstances under which such clonic movements take place is sufficient.

The behavior of the person who by accident drops a fragile object from his hands is quite characteristic. Following the momentary tonic spasm of the entire body musculature, the person executes a few movements of partial flexion and extension of the upper limbs at the wrists, elbows and shoulders. The momentary general rigidity which immediately ensues when a person has received even a slight burn is always followed by vigorous movements, which frequently involve the entire body and all the extremities. The trembling, and the movements of larger oscillation, following the rigid state in the case of exposure to the powerful emotional stimuli which bring about the states of fear and anger are always prominent. Darwin⁶⁰ describes the state of fear as follows:

“The word ‘fear’ seems to be derived from what is sudden and dangerous (H. Wedgwood; Dictionary of English Etymology); and that of terror from the trembling of the vocal organs and body. . . . Fear is often preceded by astonishment, and is so far akin to it, that both lead to the senses of sight and hearing being instantly aroused. In both cases the eyes and mouth are widely opened, and the eyebrows raised. The frightened man at first stands like a statue, motionless and breathless. . . . The mouth becomes dry, and is often opened and shut. One of the best marked symptoms is the

60. Darwin, Charles: The Expression of the Emotions in Man and Animals, 1873, p. 289.

trembling of all the muscles of the body. . . . As fear increases into an agony of terror, we behold, as under all violent emotions, diversified results. The heart beats wildly. . . . There is a death-like pallor; the breathing is labored; the wings of the nostrils are widely dilated; there is a gasping and convulsive motion of the lips, a tremor on the hollow cheek, a gulping and catching of the throat; the uncovered and protruding eyeballs are fixed or they may roll restlessly from side to side. The pupils are said to be widely dilated. All the muscles of the body may become rigid, or may be thrown into convulsive movements. The hands are alternately clenched and opened, often with a twitching movement"

The sudden desire to urinate, of the frightened or intensely surprised person, is proverbial. Urinary incontinence in women is not at all uncommon under such circumstances. The writer once had a housedog who urinated immediately and involuntarily each time he was threatened with punishment.

In the face of the familiar facts just mentioned, it will not perhaps be altogether far-fetched to call attention to the similarity of the muscular state of an attentive and "appreciative" audience and that of the muscles in the epileptic paroxysm. The muscular tension of an attentive audience is manifested by the rigid immobility of the individuals, as may be judged by the tense facial expressions and the fixed postures. It is a fact well known to political economists and historians that all social customs have their roots in physiologic and biologic phenomena. The custom of applause of an appreciative audience may not improbably have its origin in the physiologic redistribution of muscle tonus following its massive and equal distribution, the latter, in turn, being consequent on that suspension of a large portion of the cerebral functions which is implied in the act of concentration.

4. *The Stage of Recovery.*—The process of integration of the disintegrated cerebral functions in the last stage of the epileptic fit is marked by hallucinations, and the muscular reactions to these, similar to those of the first stage. Owing to the concomitant exhaustion, however, this stage is of longer duration than the first. Even when the first stage of a major attack of epilepsy is so brief that all subjective and objective manifestations of the progress of extinction of the cerebral functions are apparently absent, or when the aura is only momentary in duration, the last stage, that of recovery, is always of considerable length. The symptoms of the gradual restoration of the extinguished cerebral functions may in some cases be entirely masked by the ensuing exhaustion, and the process of integration take place during the profound sleep which follows, so that on awakening the patient may be merely dazed and stupid. In other cases, however,

when the exhaustion is not great, the gradual recovery of cerebral functions gives rise to those periods of massive supply of tonus alternating with periods of its redistribution, which are manifested in the so-called hysteroid convulsions. In still lighter attacks, automatism of a complicated kind makes its appearance. In one of the patients from the Vanderbilt Clinic series the first three stages of the attack were frequently insignificant and entirely masked by the fourth:

CASE 3.—Mary L., aged 34, a Lithuanian was housemaid in an American family. She spoke good English and the chief complaint was of times when she appeared not to understand a word of English; and, in spite of the protests of her mistress, kept up an incessant jabber in her strange tongue. Of this, however, the patient herself was quite unconscious. One such attack occurred in a crowded street while she was walking with her brother. She carried a heavy hand-bag. When the brother noticed that she was behaving in a queer manner, he attempted to relieve her. She refused to part with the bag and continued to carry it for a number of minutes until she had recovered. She was unconscious of the entire procedure. On examination it was found that the attacks of automatism were always preceded by slight motor symptoms.

In the experiments spoken of in the foregoing sections, the first stage of reduction in the conscious state was not observed; the second, that of general rigidity, though very distinct, was exceedingly brief; the third, that of clonic contraction, was insignificant; but the fourth, though not as prominent as the second, was of sufficient duration to make its observation possible in a number of instances. It lasted for about a minute, during which the subject was plainly dazed and stupid and frequently executed a number of irrelevant coordinated movements.

In case of the more powerful sudden emotional or painful stimuli, the stage of exhaustion and recovery is always very pronounced. Everybody has seen women drop into the first chair, apparently exhausted, after the spasm of general rigidity ensuing on fright, or on the reception of a very bad or even of a very good piece of news. After exposure to the stimuli which induce the severer states of fright or anger, the exhaustion may be of many hours' duration, and automatism of a complicated kind may mark the recovery of the disintegrated cerebral functions. In such cases the attack may subsequently be repeated and thus enter the domain of true epilepsy, as in the following case, which came under the writer's observation in the Neurological Clinic of the Johns Hopkins Hospital, in the summer of 1914:

CASE 4.—A healthy man, 33 years of age, complained of short lapses of consciousness on several occasions, extending over a period of some years. The last attack which was unusually long, impelled him to seek medical advice. Its history was: About a week before his appearance at the clinic, he went to a country store to procure supplies for his farm. He placed the purchases in his car. The next event of which he was conscious was sitting up in bed, a cup of coffee in his hand, and his wife beside him. The history of the interval,

of which he was unconscious, was obtained from the wife. Having loaded the car with supplies, he had wandered away into the woods where he had been discovered after a prolonged search. He was brought home and put to bed. He asked for food and drink and when it was given him, ate and drank in an apparently normal way. All the time, however, he appeared to be quite unconscious of his surroundings. The writer does not remember the total duration of the patient's unconscious state.

The history of the first attack is as follows: The patient was busy in the farmyard when someone called him to come at once into the house. He found the family crowding in a state of great alarm into the kitchen. There he discovered his father lying in a pool of blood, his throat cut and in convulsions. The young man said not a word and returned to the farmyard. A few minutes later he was discovered bothering about an old wagon. He had been quite unconscious from the moment that he saw his father dying.

The reason for the repetition of the paroxysm in such cases will be discussed later.

Instances in which the normal reaction to sudden painful or emotional stimuli is of such intensity that it cannot be distinguished from the classical major epileptic fit may be found throughout the extensive literature on epilepsy. A single example from Dr. Borie's⁶¹ cases, collected in the innocence of the early part of the last century, will suffice as an illustration:

An English army officer fought a duel with a Parisian student. The latter was the first to fire. The bullet passed through the air at some distance from the Englishman's right ear. The officer immediately fell down in a "frightful attack of epilepsy."

THE BIOLOGIC AND PHYSIOLOGIC SIGNIFICANCE OF THE NORMAL EPILEPTOID REACTION

The manner in which, among a crowd of sensory impressions, a single one succeeds in dominating the sphere of consciousness as a sensation, frequently to the utter exclusion of all other impressions, is not entirely understood.

What is true of simple sensory impressions is likewise true of the emotions and of the processes of thought. A mother who sees her child in danger of being injured is for the time being relieved of any grief with which she may be afflicted. A person intent on the solution of a difficult scientific problem may be "thoughtless" regarding his social duties.

Moreover, sensory impressions, emotions, and processes of thought are in this respect interchangeable. Instances exemplifying the manner in which an emotion, by becoming dominant, shuts out the sensations of acute pain, have been mentioned in the previous section. The

61. Borie: *Des maladies nerveuses en général, de l'épilepsie en particulier*, 1830, p. 324.

reverse is likewise true. The sensation of a severe toothache may lift entirely the burden of an emotion. And a toothache or a sudden emotion may make it impossible for the scientist or the business man to continue a train of thought.

That a relation similar to that which exists between the processes involved in the sensations, emotions and thoughts, obtains also between all of these cerebral functions on the one hand and the purely automatic functions of the midbrain and the spinal cord on the other, we have already seen. Any one who is not specially trained, and who will attempt the experiment of inducing in himself a state of general muscular rigidity by conscious effort, will discover its inadequacy as compared to the action of even such a trivial stimulus as the sudden prick of a pin. The following experiment illustrates the incompatibility of the higher cerebral functions with the tonic function of the midbrain and the spinal cord:

An old friend of the writer's, Professor C. of Washington, D. C., a frail man near seventy, was fond of exhibiting a feat of muscular prowess which none of his younger and by far the stronger friends could achieve. With the back of his head and the heels of his feet supported on the edges of two chairs he would lie straight and stiff, with as many men sitting on him as his length permitted, for a number of seconds. His eyes during this performance were tightly closed and the facial musculature tense. In order to test his conscious processes during this state of rigidity, the writer once attempted to engage him in conversation on a subject to which he was particularly susceptible. He immediately shouted "Get off!" and dropped to the floor. Upon inquiry he admitted freely that any appeal to his intellectual processes was quite incompatible with the muscular performance.

From a biologic standpoint the importance of the dominance of certain nerve functions at the expense of others must be obvious. Of the large number of vague impressions which a person experiences while tramping idly through the woods, it is obviously of the utmost importance that the sensations or emotions experienced in the presence of a snake should become dominant to the exclusion of the sensations and emotions experienced in the presence of fruit or flowers. In the long course of natural selection those animals whose nervous systems were defective in this respect must have perished, both because they could not detect and obtain their food and because they could not escape from their enemies, with the necessary degree of efficiency and dispatch.

We have seen under the heading of "Anatomic and Physiologic Considerations," that since animals are constructed on the plan of a number of jointed segments, the measured movement of a distal segment is conditioned by the fixation of the joints of the relatively central segments of the body or the limbs. When an animal is exposed to the sudden action of a stimulus, it is of the utmost importance that every

relatively distal segment be in readiness to execute movement, as soon as the kind of movement and the segments that are to participate in it, are determined by an appraisal of the nature of the stimulus. Such determination of the nature of the movement by an appraisal of the stimulus is a function of time, and is therefore a variable; while the necessary preliminary to the possible needed *movement of any kind*, is a constant, and is not conditioned by the factor of time. The act preparatory to *any* needed movement of *any* relatively distal segment consists in the fixation of *every* relatively central joint. The entire organism is thus converted into a solid base from which a measured pull can be exerted in any direction. And this is the state of the general tonic rigidity into which the organism bounds the instant it is exposed to the action of a stimulus whose nature is, for the time being, undetermined. That this state of rigidity is simultaneous with a suspension of certain cerebral activities we have already seen.

So overshadowing are, on the whole, the benefits derived by the organism from this reciprocity between the conscious state and the function of tonus, that it has become established as a rather fixed mechanism. The latter may be inferred from the fact that in a function whose goal is obviously a temporary partial suspension of all neuromuscular activity, the initial stage, which is that of suspension of the higher cerebral activities, is nevertheless accompanied by a certain amount of muscular rigidity and frequently by clonic contractions. The function in question is that of sleep and will be considered later.

The normal paroxysm described in the foregoing pages may be observed throughout the vertebrate and the invertebrate world. It may be seen in man as well as in the deer and rabbit; it may be observed in the brief movement of retraction which precedes the forward movements of escape of the surprised frog as well as in the similar movement of the frightened housefly.

It is important to bear in mind in this connection, that in the course of organic evolution a number of the primary functions of the organism come to be utilized in secondary ways, in accordance with the new needs imposed by an ever changing environment. Along these secondary ways a function may attain such a degree of development as to overshadow its primary uses. Thus, the expiratory sound which results from the contraction of the thoracic musculature in the normal paroxysms of general rigidity, comes to serve in the social animal, endowed with organs for the perception of sound, the important use of a danger signal. The cry of the frightened man, the squeak of the bird, and the croak of the surprised bullfrog all subserve this secondary purpose. Another such secondary use of the normal paroxysm of general rigidity is exemplified by the posture of rigid immobility assumed by certain insects when exposed to a danger which may be escaped by feigning death.

THE EPILEPTOID REACTION ASSOCIATED WITH CERTAIN FUNCTIONS

In the performance of every function necessitating a powerful contraction of the abdominal muscles, the entire musculature of the body and the limbs bounds into a state of increased and equally distributed tonus. This equal distribution of tonus to antagonistic muscles immobilizes the joints of every body segment and converts the entire osseomuscular mechanism into a solid base from which the abdominal muscles can exert a powerful pull. The functions of defecation, of parturition, of the expulsion of mucus or of foreign bodies from the bronchi by coughing, the acts of vomiting and sneezing all depend for their performance to a greater or less extent on the contraction of the abdominal muscles.

We have seen that an increase of muscle tonus is conditioned by the suppression, complete or partial, of certain cerebral functions. This conditioning is exemplified in a striking manner in the acts of defecation, of sneezing, and of parturition.

The circumstances under which the act of defecation is best performed by civilized adults on the one hand, and by stupid persons, children and animals on the other, point to the extent to which its performance is dependent on the relinquishment of the highest cerebral functions. It is a familiar fact that civilized adults, in the performance of this function, seek isolation. The absence of external stimuli facilitates the reduction of the conscious state down to the level where the proprioceptive impulses can exert an unhindered action on the tonic mechanism. On the other hand, young children stand in no such need of isolation in the performance of this function. In them, the cerebral inhibitory mechanism is still unripe; while stupidity is but a manifestation of poorly developed cerebral functions. Such young persons and stupid persons may be considered as possessed of the nervous system of an animal in which the lower levels are adequate to regulate the state of tonus. This is exemplified by Weed's³⁷ decerebrate young kittens, a large percentage of which failed to exhibit the phenomenon of muscular rigidity.

It is a well known fact that the epileptic fit may be averted by the action of such stimuli as may enter the sphere of consciousness in the form of sensations. Some of these stimuli have been mentioned in the introduction to this study. The same stimuli have a similar action in interrupting to a greater or less extent the progress of that reduction in the conscious state which is essential to the tonic action of the muscles in the acts of defecation, sneezing and, sometimes, parturition. The ease with which the act of sneezing is prevented by any expansion of the conscious state, as for example by a remark from a bystander on the ludicrousness of the approaching act, is familiar to all; and every

obstetrician knows how the tonic action of the muscles of the parturient woman may be interrupted by the appearance of a visitor.

The symptoms of disintegration of the highest cerebral functions during the period of transition between the waking and the sleeping state manifest themselves by the hallucinations which we call dreams. The state of awakening, which is the reverse process—that of integration—is marked by the same hallucinations. Baillarger's⁶² essay may be profitably consulted on the subject. Herbert Spencer⁶³ attempted to trace the origin of religious beliefs to the hallucinations occurring during sleep. The art of psychanalysis is utilizing these hallucinations in the discovery of the factors which determine the onset of some of the neuroses.

On the whole, authorities appear to agree respecting the activity of the muscular system during sleep. Duval's⁶⁴ statement is that with the disappearance of voluntary movement, reflex movements persist and become even easier of elicitation. Bertin⁶⁵ is certain that reflex movements are exaggerated in sleep. This investigator puts forth the remarkable idea that reflex movements would indeed occur with greater frequency in the sleeping than in the waking state if it were not for the fact that the condition of repose deprives the organism of that stimulation on which reflex movements depend. Dejerine⁶⁶ thought that the sleeping person is like the decerebrate animal; that spontaneous movement disappears, but that reflex movements persist and are perhaps elicited with greater ease. Graham Brown,²⁰ indeed, likens the state of decerebration to that of light sleep. Waller,⁶⁷ however, calls attention to the fact that in profound sleep the muscles are completely relaxed and the tendon reflexes suppressed. Rosenbach,⁶⁸ who studied the subject of sleep on young children, gives the result of his observations about as follows: In the beginning of sleep the child has a frowning expression of irritability, and the reflex susceptibility is increased. Somewhat later the reflexes are diminished, except the abdominal and the cremasteric reflexes, which persist. When fully asleep, the corneal reflex is absent, the pupils become contracted, the abdominal and the tendon reflexes are abolished.

The slight discrepancies in the conclusions of different observers are undoubtedly due to the fact that they were drawn from observations of different stages of sleep. The writer's own observations agree with

62. Baillarger, M. J.: *Des hallucinations, etc.*, Paris, 1846, p. 476.

63. Spencer, Herbert: *Principles of Sociology*, Vol. 1, 1910.

64. Duval, M.: *Sommeil*, Dictionnaire Jaccoud, 1882.

65. Bertin, E.: *Sommeil*, Dictionnaire Dechambre, 1881, p. 266.

66. Dejerine, J.: *Traité de pathologie général de Bouchard*, 5:377.

67. Waller, A. D.: *An Introduction to Human Physiology*, 1896, p. 571.

68. Rosenbach, O.: *Das Verhalten der Reflexe bei Schlafenden*, Zeitschr. f. klin. Med. 1:358, 1880.

those of Rosenbach, with the exception of a phenomenon which occurs in a still earlier stage of sleep, which is not described by that investigator. The phenomenon in question may be observed with ease on a travelling companion who, in a sitting and uncomfortable position, begins to nod. The strain of the position as well as the process of nodding maintain such a person for a considerable length of time in a very early stage of the process of falling asleep. The facial expression of such a person is tense, drawn, or frowning, and agrees with Rosenbach's description of the child's expression in the early stage of sleep. The muscles of the limbs are undoubtedly spastic. That the spasticity is not due to the strained position may be ascertained in a child by permitting it to fall asleep in the observer's arms, or as a bedfellow. On one's dog the same observations may be carried out with ease. In any such observation on persons it is important not to arouse the subject's suspicion that he is being felt with any special end in view. for the result of such a suspicion is a state of awareness, which is certain to spoil the experiment. Dogs in the first stage of sleep, may be handled by their masters with a remarkable degree of freedom without being especially disturbed, and in such animals the observations may be carried out with a high degree of certainty.

Following this initial spasticity, the duration of which depends on the rapidity of the progress of sleep, a longer period succeeds during which clonic contractions are exceedingly common. These have occurred in every person of whom the writer has made inquiries, and in every such person they have been at times of sufficient strength to awaken him or her from sleep. This phenomenon is an example of the adequacy of the stimulus of movement in aborting the progress of the normal reaction. Such clonic contractions constitute in certain instances a serious obstacle to the function of sleep and cases of this kind have been described by Gowers.⁶⁹

IV. THE PERIODICITY OF THE NORMAL EPILEPTOID REACTION

The periodicity of the epileptoid phenomena described in the last section is self-evident, such phenomena being associated with the performance of functions of a more or less regularly periodic nature.

The periodicity of those epileptoid paroxysms which have been described as reactions to external conditions, however, is not self-evident. The external conditions which evoke this reaction in the organism are purely accidental. Fright, anger, the prick of a pin, the blow of a hammer on the nail, the slamming of a door, a sudden flash of light, a burn received, and a thousand other conditions of daily life, which necessitate sudden movement or sudden attention on the part of the organism, all or nearly all belong to the category of circumstances whose occurrence it is impossible to foretell.

69. Gowers, Sir William R.: *The Borderland of Epilepsy*, p. 106.

Notwithstanding, however, the impossibility of foretelling any particular one of those conditions, their total number is so great that the occurrence of some one of them, though not of any particular one, may be foretold with certainty in the course of a given reasonable length of time. From a mathematical standpoint, therefore, the external conditions in question may, in their totality, be considered as a *constant* factor. The action of this constant factor on the organism is not always adequate to evoke an epileptoid reaction of sufficient intensity to make its presence readily observable. The reason for this variable effect of the action of the constant external factor is the changing state of the organism; and the particular state of the organism which is the most favorable for the occurrence of a pronounced epileptoid reaction is subject to more or less regular periods of recurrence.

It is a fact too well known to need elaboration in this place, that the several functions of the neuromuscular system, like all the other functions of the body, are subject to more or less regular periods of exaltation and depression; periods, for instance, when the threshold value to external impressions is lowered, alternating with periods of higher threshold values; periods of exaltation of the functions of memory, reason and judgment, alternating with periods of mental dullness; and so on. Some of these periods, either of exaltation or of depression, of certain nervous functions go hand in hand with certain other periodic functions of the body, of which the menstrual period in women may be taken as an example. Others again are the common resultants of the periodic alterations in the activity of a number of functions of the body, such as those of digestion, excretion, sleep, the sexual function, and others.

The entire subject will perhaps become less complicated when viewed from the standpoint of the elementary causes which are productive of the phenomena of periodicity. A consideration of these elementary causes, besides, will facilitate an understanding of the action of such sedatives and hypnotics as the bromids and phenobarbital in diminishing the frequency and severity of epileptoid and epileptic paroxysms, and of the reverse action of caffeine. A brief consideration of these causes will not, therefore, be amiss.

In a perfectly stationary medium it might be expected that a body consisting of a single point, when acted on by two forces emanating from two other points, would proceed to move in a perfectly straight line along the diagonal of the parallelogram of forces. No stationary medium, however, exists, nor does a body without dimensions. Owing to these latter circumstances the line of movement of bodies is never a straight one. The simple reason is that at every point in the progress of the moving body its relations to the forces which keep it in motion are disturbed by the great number of other forces in the medium. The line of motion is, therefore, one of continual readjustment. It is an

undulating or a wavy line, such that the outline of each larger wave is marked by smaller undulations, and the outline of each of the smaller undulations is indented by still smaller ones, and so on as far as they can be perceived.

In the final analysis a function of the organism is the common resultant of a great number of movements. It is to be expected, therefore, that the course of each function should be that of an undulating line, made up of phases of exaltation and depression; that each such phase should be indented by smaller phases, and these in their turn, by still smaller ones. A concrete example is the ordinary experience that at certain times of the day mental vigor is at its best. While the latter is true from day to day, it is also true that, on the whole, the mental powers are more acute during certain seasons of the year. And while this holds good from year to year, every adult has experienced the long ebb and flow of his mental faculties extending over a period of some years. And yet even this last undulation is only one which marks the outline of that large undulation of the mental faculties, the separate phases of which extend over the periods of infancy, childhood, adolescence, young adult age, middle age, senescence, and senility.

There is one more theoretical point before returning to the practical subject in hand. It must be borne in mind that the degree of undulation—the height of each phase—other circumstances being equal, depends on the *rate* of motion. Any increase in the rate, implying as it does a more difficult adjustment of the line of motion, must result in larger periodic disturbances—in swings of larger amplitude. A slower rate of movement, on the other hand, permits the resumption of the disturbed balance between the forces which keep the body in motion at shorter intervals of distance, with the result that the undulating line approaches nearer the course of a straight line.⁷⁰

In the application of this latter consideration to the concrete example given above, it will be seen that persons whose intellectual activities are normally sluggish, experience but little change in their mental function during different times of the day or during different seasons of the year. On the other hand, persons whose mental activities are intense experience these periodic changes in a marked degree.

If the course of the several functions of the nervous system is marked by periods of exaltation and depression, we can see why the constant external determining factors would be potent to bring about a pronounced epileptoid reaction only at certain times.

In the experimental production of this reaction described above, it was found to be much more pronounced in women during the menstrual period; on days when the weather was depressing than when

70. A familiar illustration is the vibration of the parts of the cheaper makes of the automobile after the machine attains a certain rate of speed.

it was bracing; and, generally, when the person was in a depressed physical or mental state than when vigorous and cheerful; and it was brought out with especial prominence under the action of a drug which intensifies the activity of the lower levels of the nervous system—under the action of caffeine, to be presently described.

V. EXPERIMENTS ON THE EFFECT OF CAFFEIN, BROMIDS AND PHENOBARBITAL ON THE EPILEPTOID REACTION AND THE EPILEPTIC PAROXYSM

The conclusion of Rivers⁷¹ and of Hollingworth⁷² regarding the action of caffeine is that under the influence of moderate doses, reactions are executed with greater despatch and correctness. Rivers, however, remarks that "when taken in excess, the stimulating action may be so transitory and followed by so great a decrease, that it may legitimately be spoken of as an accelerator of fatigue."

Hollingworth notes that, "small amounts of caffeine tend to produce retardation in discrimination time, this retardation being accompanied by a great number of false reactions. The false reactions appear to be caused by a preliminary briskness produced by the caffeine, and the retardation in the reaction time caused by a voluntary caution and the attempt to eliminate the false reactions. This is a test in which stimulation does not make for efficiency except after long practise."

The writer's experiments on the effect of this drug on the epileptoid reaction and the epileptic paroxysm, while corroborating the conclusions of the investigators just mentioned, have brought out some additional points. These experiments were undertaken with a view to determining whether the normal epileptoid reaction is essentially of the same nature, of similar origin, and subject to similar changes under similar conditions, as the epileptic paroxysm. In the approach to the solution of this problem an attempt was made to find an answer to the following questions:

1. What is the most obvious effect of caffeine and of the sedatives on a vertebrate organism whose nervous system may be considered to correspond to the lower levels of the human nervous system?
2. What is their effect on the functions of the different levels of the normal human nervous system?
3. What is their effect on the subnormal nervous system of the epileptic?

In the choice of vertebrates of low organization the advantages offered by those having a smooth, hairless, linear body without appendages are obvious. Such animals are snakes.

71. Rivers, W. H. R.: *The Influence of Alcohol and Other Drugs on Fatigue*, Croonian Lectures, 1906, p. 50.

72. Hollingworth, H. L.: *The Influence of Caffeine on Mental and Motor Efficiency*, *Arch. Psychol.* **3**: No. 22, 1912.

EXPERIMENTS ON REPTILES

The experiments⁷³ were conducted on black snakes and on garter snakes, altogether fifteen in number. The results were quite uniform and the description of a single experiment will apply to all the others.

Six snakes were fastened by strips of adhesive plaster to a table parallel to each other and at some distance apart. One piece of the strip was passed around the neck, another midway between the two ends and the third about five inches from the aboral end. Two snakes were injected with sodium bromid, $\frac{1}{4}$ grain (0.015 gm.) and $\frac{1}{2}$ grain (0.03 gm.) respectively to the pound of body weight; two with the same amounts of caffein citrate, and two were left for control. Ten minutes later the six reptiles all lay quietly without exhibiting any tendency to struggle or to move in any way. The table was then gently tapped once with the finger. The bromidized and the normal snakes remained perfectly quiet, while the two caffeinized snakes exhibited a remarkable reaction. It was a wave-like movement whose outline was marked by a vibration which passed with great rapidity from head to tail. The duration of the entire reaction was about half a second; it then subsided completely. A stronger tap on the table produced the same reaction in a more pronounced form—the amplitude of the movement was greater. The reaction was more pronounced in the reptile which had received the larger dose. By repeated tapping of the table one played on the two reptiles as on a stringed instrument, except that the vibrations in this case were not audible but visible.

When touched gently near the tail end, the control and the bromidized snakes made no response, while the caffeinized snakes responded by a brisk movement. A stronger touch brought out some movement in the normal snakes. The same movement in the bromidized snakes, though of smaller amplitude, was of somewhat longer duration; and the movement was most pronounced and briefest in the caffeinized reptiles.

The experiments on these animals with other drugs and after mutilation, are not relevant to the subject in hand and will not, therefore, be discussed.

The obvious answer to the first question, then, is that caffein enhances to a very marked degree the capacity of the lower levels of the central nervous system for the reception of the simpler and more common kinds of stimuli and the response to these by simple reactions. The action of the bromids is the reverse of that of caffein.

SUBJECTIVE EXPERIMENTS

The question respecting the effect of these drugs on the functions of the several levels of the central nervous system seemed difficult to answer from purely objective experiments. The writer, therefore, undertook to experiment on himself, with the following results:

The action of caffein in more than moderate doses (caffein citrate 15 grains [1.0 gm.] three times a day, in addition to three cups of

73. The writer wishes to express in this place his gratitude to the administration of the New York Zoological Gardens, and especially to Mr. Ditmars, the curator of reptiles, and to Mr. Moony, the keeper, for their liberality and courtesy. The abundance of material and the amount of assistance which these gentlemen placed at the disposal of the writer left nothing to be desired.

coffee) was to enhance to a very marked degree the susceptibility to all simple impressions, and the response to such impressions by simple automatic reactions. The epileptoid reaction manifested itself in the author in a striking manner on exposure to kinds of stimuli which had never evoked this reaction before. The sound of shutting a door, the unexpected sight of an acquaintance, the momentary idea that an object resting on the edge of a table might drop to the floor, and any of the numerous conditions which cause the sudden arrest of attention, or which demand sudden movement, brought on a momentary state of general rigidity, and a subsequent period of exhaustion and "bewilderment." The typewriter was operated with greater facility and despatch, the exercise of handwriting was made more rapid, and so were all acquired automatic movements. While reading was much faster, the contents did not ingrain themselves with the necessary clearness. The coherence of a train of thought was very markedly diminished, its elements succeeding each other with great rapidity. After four days the matter read appeared devoid of meaning; coherent thinking became very difficult and a state of excessive alertness and hyposomnia set in, which necessitated the discontinuance of the drug.

The action of bromids (30 grains [2 gm.], three times a day) was in certain respects the reverse of that of caffeine. The capacity for the perception of simple impressions was markedly blunted, and all simple reactions became sluggish. During the six days when the drug was taken manifestations of an epileptoid reaction were not once perceived. Reading became difficult and the matter read left a hazy, indistinct impression. Thinking, although sluggish and difficult, was not, however, marked by the incoherence which had made the process defective when caffeine was taken.

The action of phenobarbital was, on the whole, the same as that of the bromids. It appeared to the author, however, in his subjective experiments, that phenobarbital (3 grains [0.2 gm.] four times a day for four days) had, in addition, a rather disagreeable effect on the circulatory system, as judged from slight cyanosis of the finger tips and shortness of breath on exertion. The latter action, however, may have been due to a special susceptibility.

After taking caffeine, the highest integrative nerve functions failed for two reasons. One was on account of the unusual crowding of external impressions. The other was because attention was continually distracted by involuntary reaction to each impression in the crowd. The distinguishing feature of the highest functions of the nervous system is a response by a single reaction to an accumulated number of separate impressions, after the latter have been integrated in a definite manner. The immediate response to each separate stimulus received was, therefore, in itself incompatible with these highest activities.

The reason for the action of the bromids and of phenobarbital in diminishing the capacity of the highest nerve functions, on the other hand, appeared to be the tardiness with which impressions followed each other, the paucity of those which entered the domain of consciousness as definite sensations and emotions.

THE EFFECT OF CAFFEIN AND PHENOBARBITAL ON THE EPILEPTIC MANIFESTATIONS

As the most pronounced effect of caffein was to produce a wide-awake state of the kind described, it appeared that the epileptoid reaction manifested must be essentially different from the epileptic paroxysm. The muscular phenomena of the first appeared to be brought about merely by an increased alertness; those of the second are undoubtedly preceded by a suspension or reduction of the conscious state. The experimental administration of caffein to epileptics was, therefore, approached with a sense of the possibility of substituting a pronounced normal epileptoid reaction for the epileptic paroxysms. The doses of caffein given were, however, small, the largest being 3 grains (0.2 gm.) four times a day, the smallest 1 grain (0.06 gm.) at bedtime. The cases included every possible category of the epileptic disease: organic as well as nonorganic, inherited and noninherited, major, minor and psychic. The following ten cases, picked out at random, give a fair idea of the effect of the three drugs on the disease:

CASE 5.—J. P., a boy, aged 17, of robust, healthy appearance, presented no apparent stigmata. Birth, infancy and childhood had been normal and he had been normal at school. No familial history of epilepsy was elicited. Five months before his appearance at the clinic, he struck his head against an iron banister and was unconscious for twenty minutes. The scalp was not bruised. A few hours later there occurred a major epileptic fit, the first he had ever had. Two days later there was a second major fit and after that fits occurred at irregular intervals, on the average five a week. He had been taking drugs for some weeks, which diminished the number and severity of the fits. Minor fits only had occurred for the last two or three weeks on the average of one a day. During these the face was drawn to the left side.

Roentgenograms of the skull were negative. The reflexes were normal and so were apparently the internal organs. The mentality was dull and tardy; memory was very poor. Relatives stated that before the onset of the attacks the boy had been very bright.

The patient was given caffein citrate, 3 grains (0.02 gm.), four times a day. Five days later he reported eleven major attacks and a great number of minor ones. Mental dulness was very much increased and confusion of ideas was so great that he had great difficulty in understanding what was said to him. Caffein was discontinued and phenobarbital, 1 grain (0.06 gm.), three times a day, was given instead. A week later great improvement was reported. No attacks had occurred and mental dulness had to a great extent disappeared. In the next three weeks the patient, still taking phenobarbital, reported one minor attack.

CASE 6.—A. P., a girl, aged 6, had the appearance of a normal child, except for abnormally long ear lobules, inherited from her mother. There had been a difficult instrumental delivery, but development was normal. Minor fits had been observed for the last eighteen months on an average of once a day. No hereditary history of epilepsy was elicited. The tendon reflexes were slightly increased on the left side; there was no Babinski sign, but the right toe went down with greater constancy than did the left on plantar stimulation. The abdominal reflexes were active and equal.

The child was given phenobarbital, $\frac{1}{4}$ grain (0.015 gm.) once a day, which reduced the number of fits to one in two days. Two weeks later, caffein citrate, $\frac{1}{2}$ grain (0.03 gm.) was given three times a day. Eight days later it was reported that she had had one or two fits every day since last seen. The child was again given phenobarbital, $\frac{1}{4}$ grain (0.015 gm.) twice a day, with the result that the fits occurred once in two days.

CASE 7.—Wm. A., a lad, aged 18, gave a history of normal birth; he had been delicate as a child, but development was normal. At the age of 6 he fell from a swing and struck his head badly, but there were no immediate serious consequences. The record of his school career was very poor. In the last two years he has had altogether two or three major attacks. Minor attacks have occurred on the average twice a week. The minor attacks consist at times merely in a "sensation" or feeling of dizziness. A niece of the patient has *epilepsy and is feeble minded.

Examination of the labyrinths made by Dr. Rosenbluth, disclosed no abnormality. The reflexes and internal organs were normal. The mentality was dull and sluggish.

The patient was given phenobarbital. Two weeks later he reported one attack of dizziness and one "sensation"; no loss of consciousness. Phenobarbital was discontinued and caffein citrate given instead, 1 grain (0.06 gm.) every four hours. Eight days later he was reported as feeling generally brighter in mind. Only one attack of dizziness had occurred. Within the next two weeks there were a very great number of attacks of dizziness (exact number not known). Caffein was discontinued and phenobarbital given instead. A week later he was reported as having had no attacks. The mental condition was as dull as before caffein had been taken.

CASE 8.—S. R., a woman, aged 27, had a definite facial asymmetry, the left side being fuller and sagging. The left palpebral fissure was larger than the right. There was no history of epilepsy or insanity in the family.

Major epileptic attacks had occurred since infancy. At the age of 14 these became much more pronounced and occurred about once a week. She had not menstruated until the age of 24, when she came to the Vanderbilt Clinic. There, she was placed first on bromids, then on phenobarbital. She menstruated soon after. Attacks after that recurred once a month, during the menstrual period.

The reflexes were normal. She could not voluntarily turn the mouth to the left, nor could she close the left eye without at the same time closing the right.

She was given caffein citrate, 3 grains (0.2 gm.) four times a day. Three major fits took place within the next four days. Caffein was discontinued and phenobarbital was given again. Within the next month she had one attack, during the menstrual period, as before.

CASE 9.—L. S., a woman, aged 44, of normal appearance, gave no direct hereditary history of epilepsy. Nocturnal attacks of major epilepsy appeared five years before, without known cause, and had recurred on the average of one every eight months. Attacks of motor aphasia of about one hour's duration had also been observed on the average once a month, for the last five

years. For the last two or three years the right upper limb had become paralyzed for a few minutes nearly every day. Of late the left arm, too, had become affected in the same way. She complained of failure of memory and of difficulty in thinking.

She had been taking bromids and phenobarbital for the preceding three years; there had been no major attack within the last twelve months.

The blood Wassermann reaction and roentgenograms were negative. The right tendon reflexes were somewhat exaggerated.

The patient was given caffein citrate, 3 grains (0.2 gm.) four times a day. A week later she reported as feeling generally brighter and more cheerful. She had not the "dopy" feeling that she had before caffein was given, but had had difficulty in falling asleep. Caffein, 3 grains (0.2 gm.) was then given once a day only. Three days later she had eight major attacks in one night. She was unconscious all night and most of the following day. Caffein was discontinued and phenobarbital therapy again instituted. A month later she reported herself as feeling the same as before caffein had been taken. There had been no major attacks.

CASE 10.—F. S., a man, aged 30, except for a slight facial asymmetry, appeared normal. He had a hypospadias. There was no history of epilepsy in the family.

Eight years before, the patient had been for a time under unusual stress, working all day and attending on his invalid mother at night. One night, when especially fagged out, he had a nocturnal major fit, which had been repeated on the average once every six months until eighteen months before. For the last eighteen months he had been taking drugs regularly and had not had any fits. He complained of mental dulness and sluggishness and of a feeling of great fatigue on the least exertion. The reflexes and internal organs were normal.

Phenobarbital therapy was discontinued and caffein citrate, 3 grains (0.2 gm.) four times a day, was given instead. For the next ten weeks he reported weekly. A great improvement was noted in his general condition. The mental dulness and sense of fatigue disappeared and he became bright and cheerful. For the first time in his life he felt like a "real man." An occasional momentary feeling of dizziness was experienced, which caused no discomfort. A singular change took place in the patient's character. He was no longer the same modest young man as before caffein was given; he was boastful and vainglorious. The caffein was reduced to 3 grains (0.2 gm.) once a day, given early in the afternoon.

CASE 11.—R. R., a woman, aged 18, appeared normal. At the age of 5 months a series of convulsions extending over a period of three days was observed. Otherwise infancy and childhood were normal. There was no history of epilepsy or of insanity in the family.

Minor attacks had occurred on the average of one a day, for the last three years. She had been taking phenobarbital for about twelve months, and during that time, had had three or four attacks during menstrual periods only.

The reflexes and internal organs were normal. The mental status was normal to ordinary tests. She had an insuperable dislike for any kind of mental effort and had no intellectual interests of any sort. She could not bear to read, or even to see a movie. On the contrary, she had a great passion for manual labor, and worked rapidly and indefatigably from morning to night and enjoyed it. She had a quick and violent temper.

The patient was given caffein citrate, 3 grains (0.2 gm.) four times a day. Within the next five days she had twenty-six minor attacks. Caffein was discontinued and phenobarbital given instead.

CASE 12.—H. L., a man, aged 22, appeared normal except for a large and very ugly nose. Of thirty-two known blood relatives, not one had epilepsy. Infancy and childhood were normal.

Major and minor attacks had been observed for the last six years. During the first three years the major attacks recurred about once a week but after that time he had taken drugs regularly and had not had any major attacks for a number of months. Minor attacks took place on the average of one in two days, during which there was marked automatism. The reflexes and internal organs were normal.

Phenobarbital therapy was given for seven weeks. In this period there were no major attacks and only four minor ones weekly. He was given caffein citrate, 3 grains (0.2 gm.) four times a day. A week later he reported that he had had six minor attacks. Within the following five days the patient had three major and a number of minor attacks and he appeared demented at times. Caffein was discontinued and phenobarbital given once more.

CASE 13.—R. W., a man, aged 22, appeared healthy and well built. He had a facial asymmetry and large, congested hands. Both traits were inherited from the mother's side of the family. An aunt on the mother's side and a grandmother both had epilepsy.

Birth, infancy and childhood were normal, but he had a rather poor school career. About three years before he began to have nocturnal major attacks, without definite determining cause. The attacks were repeated about once a month. The reflexes and internal organs were normal.

The patient was given phenobarbital and the attacks ceased. Caffein citrate was then given. A week later he reported two nocturnal attacks and complained of irritability and restlessness. Caffein was discontinued and phenobarbital therapy instituted. No attacks occurred during the next two months.

CASE 14.—G. W., a boy, aged 11, appeared normal, except for a notable difference in the size of the two ears, the left being the larger. The latter characteristic was inherited from the father. No epilepsy or insanity was discovered in the family. The father, however, suffered from "nervousness" to such an extent that he had had to abandon his occupation.

Birth, infancy and childhood were normal. Minor epileptic attacks had been observed for the last three years. The attack sometimes amounted only to a "sensation." At other times, however, the right arm was raised over the head and became very rigid; at the same time the patient's face turned to the right, and his body rotated in the same direction. At still other times, the attack consisted in automatic coordinated movements, such as running about the room, etc. The reflexes were normal and the internal organs healthy.

The patient had been taking phenobarbital for some months, with the result that the attacks had become very light and recurred about once a week. He was placed on caffein citrate, 1 grain (0.06 gm.) three times a day. Seven days later he was reported as having had four very severe attacks. Two of these were major—the first major attacks he had had. The drug was immediately discontinued and the patient was once more placed on phenobarbital.

The action of these drugs may now be summed up in the most general terms:

It will be remembered that the amplitude of oscillations in a line of motion depends on the rate of the motion. In terms of force, therefore, the action of caffein was to increase the amplitude of oscillations of the course of certain nerve functions by an acceleration of the rate

of their motion. The bromids and phenobarbital, on the other hand, by retarding the rate of the movement, diminished the amplitude of oscillation of those functions and made their course nearer that of a straight line. Exogenous and endogenous stimuli, therefore, were at an advantage in evoking reactions in the first instance and at a disadvantage in the second. A chronic state of sluggishness of nerve function induced by the action of the bromids or phenobarbital is the price paid by the epileptic person for his rescue from the danger of a paroxysm.

VI. THE RECIPROCITY OF NERVE FUNCTIONS. THE IDENTITY OF THE EPILEPTIC PAROXYSM WITH THE NORMAL EPILEPTOID REACTION

The reciprocal mechanism for the innervation of antagonistic muscles has been dealt with in a previous section. We have seen, too, that a similar reciprocal action is to be found at higher levels of the central nervous system. Prominent among the latter are the tonic centers of the spinal cord and midbrain on the one hand and of the cerebrospinal and cerebropontine systems on the other. At still higher levels, we found that a similar reciprocity exists in the domain of the sensations, the emotions and of thought.

If, now, we make a general division of the nerve structures into low—those which receive impressions and respond to them by simple immediate reactions—and high—those which inhibit and integrate—we shall likewise find them to be subject to the general rule of reciprocity of nerve functions—we shall find that the active functioning of one set of structures is simultaneous with an inhibition of function in the other. The incessant restlessness of the dog or the monkey with the ablated cortex indicates that the functioning of this highest nerve structure in the intact animal is concomitant with an inhibited activity of the lower nerve structures. And the same fact is in another way exemplified by the action of large doses of caffeine on the normal human being. That action we have seen to be a great enhancement of the lower nerve structures for the reception of and the response to impressions; and such an effect we have seen to be incompatible with the exercise of the highest functions of integration and of discrimination.

Mention was made in a previous section of the fact that certain primitive and dormant mechanisms in the lower levels of the nervous system may awaken to action when the higher and more efficient mechanisms, subserving similar functions, are removed. The statement must now be amplified by calling attention to the fact that such primitive mechanisms have for their salient characteristic the ready reception of simple stimuli and the immediate response to them by muscular movements of a simple pattern. If a transient disintegration of the highest functions is accompanied by an enhancement of the lower kinds of reactions, what must we expect when the highest struc-

tures of the brain are in any way permanently defective? In that case we may not only expect the enhancement of the lower order of reactions which result from the lack of a higher control, but, in addition to that, a resurrection of a number of ancient and long dormant mechanisms in the lower levels of the nervous system. The enhancement of the lower order of reactions must then be out of all proportion to the defect of the highest structures.

The latter consideration enables us to understand the extraordinary effect of small doses of caffeine on the epileptic paroxysm. The same intensification of the lower nerve functions by caffeine which, in the normal person, results in an exaggeration of the normal epileptoid reaction must, in the case of the epileptic person, set going a series of mutually aggravating conditions. A vicious circle is created in which, with each degree of enhancement of the lower nerve functions, there is an increased disintegration of the higher; and each degree of increased disintegration of the higher functions, in its turn, brings about an enhancement of the lower order of functions. In such cases, therefore, we are prepared to find the most pronounced manifestations of the epileptoid reaction. In these patients the effect was manifested by an exaggeration of the epileptic paroxysms.

We have seen, however, that the difference between the epileptoid reaction and the epileptic paroxysm is merely one of degree of intensity. We have brought examples of cases in which the normal epileptoid reaction could in no way be distinguished, even with respect to intensity, from the epileptic paroxysm, except that in the latter case the paroxysm was repeated. The reason for such a stability of the severer form of the reaction abides in those permanently enhanced capacities of the lower levels of the nervous system, which are concomitant with some permanent defect of the highest structures. Whether the defect is inherited or acquired makes no difference. Hauptmann⁷⁴ who made a study of fifty-two patients in whom epilepsy seemed to make its appearance as a result of exposure to war conditions, was able to trace, in nearly every instance, some pre-existent defect of the highest nerve functions. In the case of the accident to the school teacher and in Case 4, we see examples of epileptoid and epileptic reactions which approach the borderline from either side. The fact that the father in Case 4 committed suicide in a particularly shocking way indicates the probability of a permanent inherited defect of nerve structure in the son, and accounts for the recurrence of the vastly exaggerated epileptoid reaction.

In about half the total number of epileptics, the paroxysms are nocturnal. In the process of falling asleep and of awakening from

74. Hauptmann, A.: Ueber Epilepsie im Lichte der Kriegserfahrungen, *Zeitschr. f. d. ges. Neurol. u. Psychiat.* **36**:181, 1917.

sleep, the nerve functions exhibit the effect of a generally depressing condition; the highest functions are to a great extent extinguished, while the lower are still active. Such a state furnishes every circumstance for the release of the mechanism involved in the epileptoid or the epileptic fit: the disintegration of the conscious state, with the consequent freedom of action of the tonic mechanisms of the midbrain and the spinal cord. In the state of awakening the highest structures are last to recover. Here, too, we have the conditions necessary for the production of the fit: the full capacity of the lower nerve functions, coincident with the still depressed high cerebral activities. We have seen that the normal process of falling asleep exhibits the epileptoid reaction. In case of a subnormal structure of the brain, therefore, the process of falling asleep and of awakening must be very favorable to the onset of an epileptic paroxysm. The strikingly frequent occurrence of the epileptic paroxysm in sleep is, therefore, in exact accord with the other facts pointing to the essential identity of the normal epileptoid reaction and the epileptic paroxysm.

The character of the pronounced epileptic is beyond a doubt abnormal. Even that enhanced docility of temperament, which is frequently hailed as a rare virtue, must, both from a neurologic and a sociologic standpoint, be considered a defect. Such extreme toleration of the vices and weaknesses of one's fellows as is displayed by Dostoyevsky's⁷⁵ Prince Mishkin, makes, in the long run, for the downward progress of the individual as well as for that of the race.

Extreme docility, however, is not incompatible with outbursts of wild rage. Case 12 possessed unusual docility and sweetness of temperament. Yet this patient complained of fits of rage and intolerance which impelled him on frequent occasions to commit violence on his fellows.

After months of study of Case 10, the writer came to the conclusion that the character of that patient was normal. After the patient had taken caffeine for two or three weeks, the writer was one day astonished to hear him break out in a stream of boastfulness and vainglory. He boasted of the size of his genitals and of his prodigious sexual powers. On investigation both claims proved to be untrue. The defect in the faculty of discrimination became manifest when the disintegration of the highest faculties, brought about by caffeine, was added to that which was inherent.

After a similarly prolonged study of the character of another patient of the Vanderbilt Clinic Series, the conclusion arrived at was that he was a model young man. He worked steadily, was devoted to his parents, spent his evenings at home and never went in bad company. He was a devout and regular attendant at church; he had never had an affair with a woman, being sincerely convinced of the

75. Dostoyevsky, F.: *The Idiot*.

sinfulness of illicit relations. One day he admitted being an habitual liar, a thief and a traitor. He was in the habit of lying on the least provocation. He had stolen money from his parents and from his relatives. He had robbed a cash register, though he did not really need the money. He had stolen an implement in his place of business. The object was soon missed and, in the patient's presence, a friend of his was accused of the theft. The result of the whole affair was that the real hero of the occasion was considered, more than ever before, the model of an honest, modest and capable employee.

The other patients yielded more readily a knowledge of the profound defects in their characters. It must be said, however, that these defects, far from being defects of instinct, as Clark¹² puts it, were rather the result of a defective inhibition of the instinct. Every one of the faults of character of these patients was obviously due to a tendency to react immediately and mechanically to environmental stimuli. They lacked that faculty of integrating impressions received, which makes itself manifest in the exercise of judgment and discrimination.

Viewing character as the sum total of the reactions of the human organism, the character of the epileptic person is closely correlated to his disease. This disease consists, as we have seen, in an enhanced capacity for the manifestation of the normal epileptoid reaction, brought about by a corresponding incapacity of the highest cerebral functions. In terms of "character," such an incapacity implies a relative inability to exercise the faculty of judgment; while the enhanced capacity for the immediate response to environmental stimuli implies, in the same terms, a release of the lower animal instincts.

Viewing epilepsy from the standpoint of the mechanism and the cause, as outlined in the foregoing pages, a rational mode of prevention and of therapy will suggest itself to the thinking physician; and this must constitute the subject of a separate publication.

VII. SUMMARY

The investigation discloses a physiologic reaction in normal man and animals which consists of the following connected train of phenomena:

1. A temporary reduction, disintegration or extinction of the cerebral functions.
2. A tonic contraction of the entire skeletal musculature, with a certain predominance of the extensors, adductors and pronators; the consequent fixation of the joints resulting in the posture characteristic of the state of decerebrate rigidity.
3. Clonic muscular contractions, i. e., alternating contractions of antagonistic groups of muscles.
4. Recovery, marked by symptoms of integration of the disintegrated cerebral functions and a state of general exhaustion.

This reaction, in a more or less pronounced form, normally occurs under two sets of conditions: (1) on exposure to the action of a stimulus requiring sudden movement on the part of the organism, or the narrow focusing of attention, and (2) in connection with certain functions, such as sleep, defecation, sneezing, coughing, parturition.

The mechanism of this reaction is to be found in certain anatomic structures and physiologic functions underlying the production and distribution of muscle tonus in the nervous system.

The biologic purpose of the reaction is the automatic fixation of the relatively central joints preparatory to any possibly needed movement of relatively distal segments of the body and limbs.

In the history of any individual organism this reaction occurs with greater frequency and in more pronounced form at certain times than at others. In other words, the reaction is characterized by a periodicity of occurrence.

The phenomena which constitute the normal reaction described, being in a great number of particulars the same as those which constitute the epileptic paroxysm, a suitable name for it would appear to be *the normal epileptoid reaction*.

The normal epileptoid reaction is elicited with greater ease under the influence of the same agencies which cause an exaggeration of the epileptic paroxysm. An example of such an agency is the action of caffeine. On the other hand, influences which are potent in holding the epileptic paroxysm in abeyance, such as the action of the bromids and phenobarbital, have the power to diminish the manifestations of the normal epileptoid reaction.

In numerous cases it is impossible to determine whether the paroxysm belongs to the category of the normal reaction or to that of the epileptic disease. Mild cases of epilepsy and instances of the normal reaction of the severer grades are in every respect alike. The normal reaction may, however, be of such severity as to be indistinguishable from the major epileptic paroxysm.

The cause of the generally greater severity of the epileptic paroxysm is to be found in a neurologic principle which underlies the production of the normal epileptoid reaction—that of the alternating reciprocity of function of the higher and lower levels of the nervous system. A slight and transient suspension of the higher functions, resulting as it does in a release of function of the lower levels, brings about the normal epileptoid reaction. Any factor, therefore, which operates permanently so as to cause large periodic suspensions of the highest nerve functions, whether that factor be a brain tumor, a poison in the blood, a peripheral irritation or an inherited defect, will, by a permanent exaggeration of the normal reaction, tend to degrade it to the clinical picture of the epileptic paroxysm.

LESIONS OF THE OPTIC CHIASM AND TRACTS WITH RELATION TO THE ADJACENT VASCULAR STRUCTURES *

TEMPLE FAY, M.D., AND FRANCIS C. GRANT, M.D.

PHILADELPHIA

It is of unusual interest to note how few gross deformities are found at necropsy to explain the variety of visual field defects caused by suprasellar and interpeduncular tumors. Pressure on the visual pathways is the generally accepted cause, but how and where it is exerted is often a matter of speculation. That only 13 per cent. of the 148 cases of tumor in this area collected by Uhthoff¹ showed gross lesions of the tracts, is significant. As pointed out by Bartels² and Cushing,³ there is usually little at necropsy to explain the complete hemianopias so often associated with these lesions.

The great destruction of the optic tracts seen in a recent case, caused by a large interpeduncular tumor, and the relationship of the offending adjacent vascular structures are of decided interest, and an attempt has been made in this case to correlate the visual field defects with the gross anatomic picture.

It is our purpose to present a case which showed deep notching of the optic tracts by the carotid arteries caused by pressure against them from an interpeduncular tumor. A search has been made for similar cases, and where diagrams existed they have been reproduced in order to throw further light, if possible, on visual field defects produced by such lesions.

There are four structures in this area which may exert direct pressure against the visual pathways in the presence of a large invading suprasellar lesion: the dural band connecting the anterior clinoid processes, the internal carotid arteries, the anterior cerebral arteries and the anterior communicating artery. The anatomic relations of these structures may be of interest.

The carotid artery, after leaving the carotid canal, passes forward through the middle lacerated foramen with the sixth nerve and the cavernous sinus, to a point just below the anterior clinoid process. Here it bends upward and backward in horseshoe fashion, to reach a position just below and to the outer side of the chiasm proper. It

* From the clinic of Dr. Charles H. Frazier, University Hospital, Philadelphia.

* Presented before the Philadelphia Neurological Society, April 28, 1922.

1. Uhthoff: Graefe-Saemisch. Handb. d. ges. Augenheilk. **11**:1262.

2. Bartels: Ueber Plattenepithelgeschwülste der Hypophysengegend (des Infundibulums), Ztschr. f. Augenheilk. **16**:407, 1906.

3. Cushing, Harvey, and Walker, Clifford B.: Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumor, Arch. Ophth. **47**:119, 1918.

then passes upward almost at a right angle, about 1 cm. behind the chiasm, almost touching the optic tracts, and quickly divides above into its terminal branches (Figs. 1 and 2).

The anterior cerebral artery passes forward over the optic tracts and chiasm as it ascends and converges toward the midline to pass up between the frontal lobes.

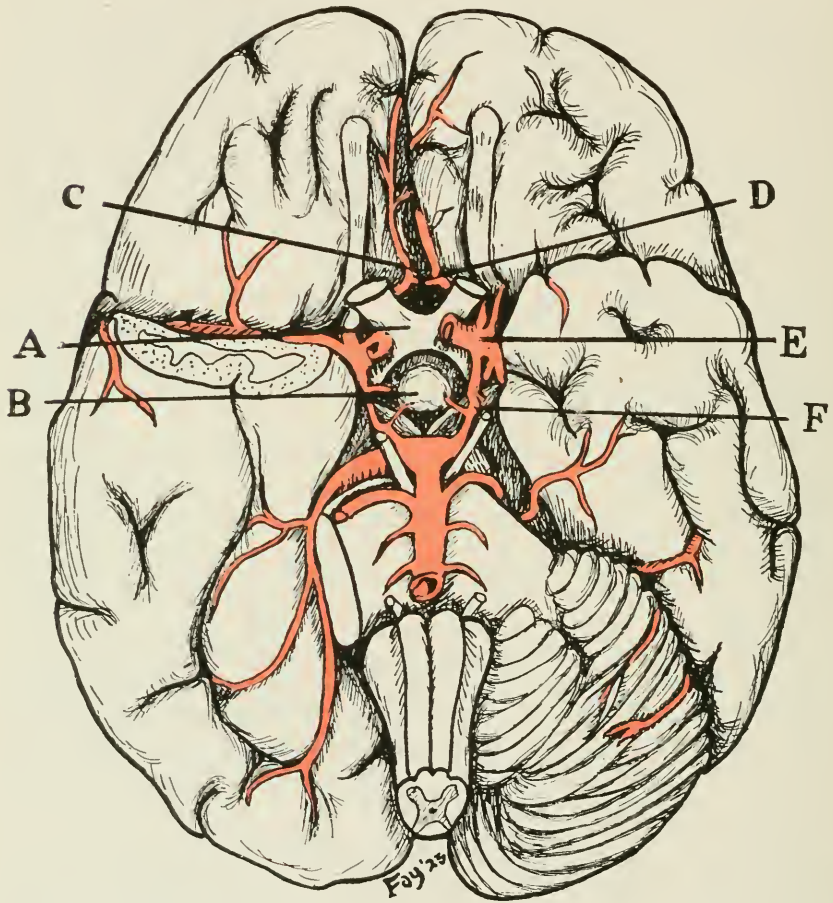


Fig. 1.—Relation of the arteries to the optic chiasm and pituitary on the base of the brain: *A*, optic chiasm; *B*, pituitary gland; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, internal carotid artery; *F*, posterior communicating artery.

The anterior communicating artery is usually given off at a point just above and in front of the chiasmal decussation. The artery itself is small and seldom over 1 cm. in length. The posterior communicating arteries flank the interpeduncular space and lie well to the outer side of and below the optic tracts, and hence produce no influence on these structures.

The vascular structures that may be concerned in the presence of interpeduncular tumors are therefore the internal carotids, the anterior cerebrals and the anterior communicating arteries. Cushing³ points out that there may be constriction of the nerve by a dural band connecting the anterior clinoid processes and cites a case of this character (Fig. 3).

HISTORY

It may be of interest to note that attention was first called to this type of lesion by Türck⁴ in 1852. He reported a case of tumor of

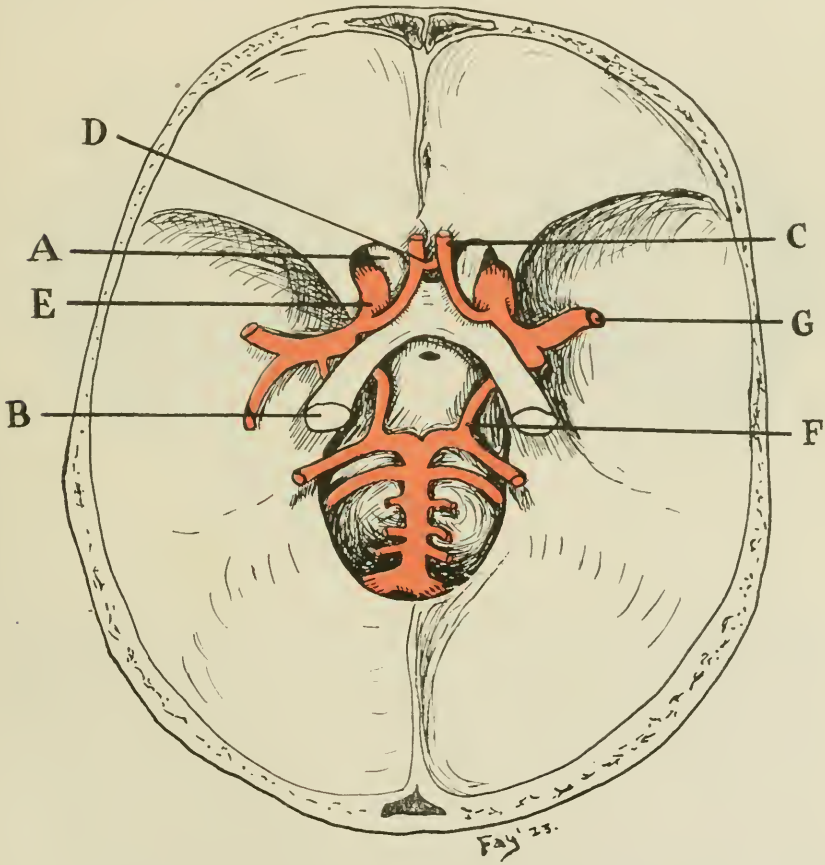


Fig. 2.—Relation of the arteries to the optic nerves, chiasm and tracts: *A*, left optic nerve; *B*, left optic tract; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, internal carotid artery; *F*, posterior communicating artery; *G*, middle cerebral artery.

the hypophysis in which both nerves were deeply notched transversely by the anterior communicating artery; and he stated that the tract was also notched laterally on the left, but does not say by what vessel. The left nerve was so deeply cut that few fibers remained intact. The

4. Türck: *Ztschr. d. k. k. Gesellsch. der Aertz. zu Wien* 8:299, 1852.

right nerve showed the inner third uninjured. There is no report concerning the fields in this case, but a note made one week before death stated that the left eye was completely amaurotic, while the right eye was only amblyopic. Sachs,⁵ in reporting the findings in forty-eight necropsies which revealed hypophyseal tumors, twice found constriction of the optic nerves by the anterior cerebral arteries. In one of his cases, the tumor presented itself in front of the chiasm. Erdheim⁶ has collected seven cases of optic tract and nerve constriction. Two cases showed the tracts almost cut in two, 1 cm. behind the chiasm,

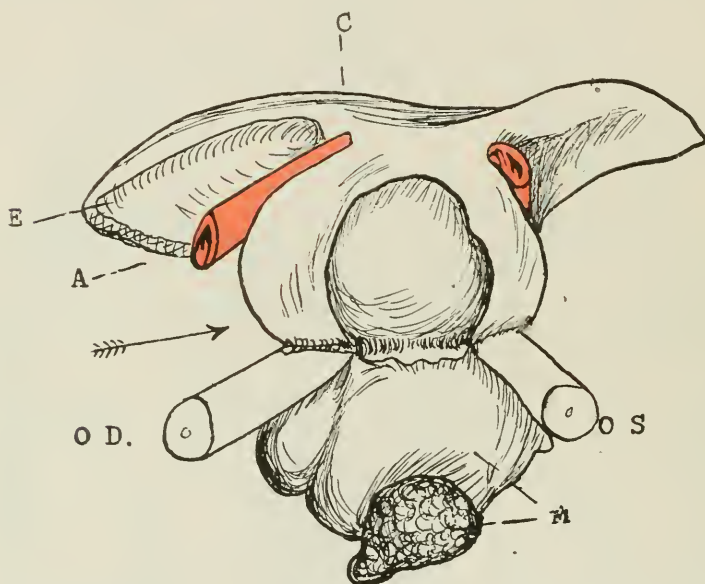


Fig. 3.—Front view (copied from the article by C. B. Walker and Harvey Cushing, *Arch. Ophth.* **47**:119, 1918) of the distortion of the optic chiasm and nerves produced by a hypophyseal adenoma which extended into the cranium. The right optic nerve is deeply cut by pressure against an artery. Both optic nerves have been compressed at the level of the arrow by a band of dura which connected the widely separated clinoid processes. *M*, the intraseal tumor mass; *C*, the chiasm on top of the tumor; *O D.* and *O S.*, the right and left optic nerves; *A*, an artery deeply embedded in the right nerve at the chiasm; *E*, extension of the tumor into the temporal lobe. The visual fields presented "left homonymous hemianopia, incomplete in the left eye, with decided central scotoma in the right eye and contraction of the lower nasal quadrant."

from pressure by the carotids. The remaining five cases showed notching of the nerve or chiasm by the anterior cerebral and anterior communicating arteries. Visual fields do not accompany the reference. Uthoff,¹ in reviewing the literature on hypophyseal disease for more

5. Sachs: *Ztschr. f. Augenheilk.* **13**:378, 1905.

6. Erdheim, cited by Bartels.

than 100 years, up to 1911, reports a case of vascular constriction of the optic nerves by the anterior cerebral arteries. The visual fields showed a bitemporal hemianopia, with contraction in the lower nasal fields. It is possible that the latter defect was caused by these arteries cutting the upper and outer tracts of the nerve which supply the lower nasal fields. Bartels,² in an extensive contribution to the pathology of this area, also reports a case in which pressure of a tumor from behind had pushed the chiasm forward, and in which the optic tracts were deeply notched by the anterior cerebral arteries. He points out that, owing to the location of the lesion, it is readily overlooked.

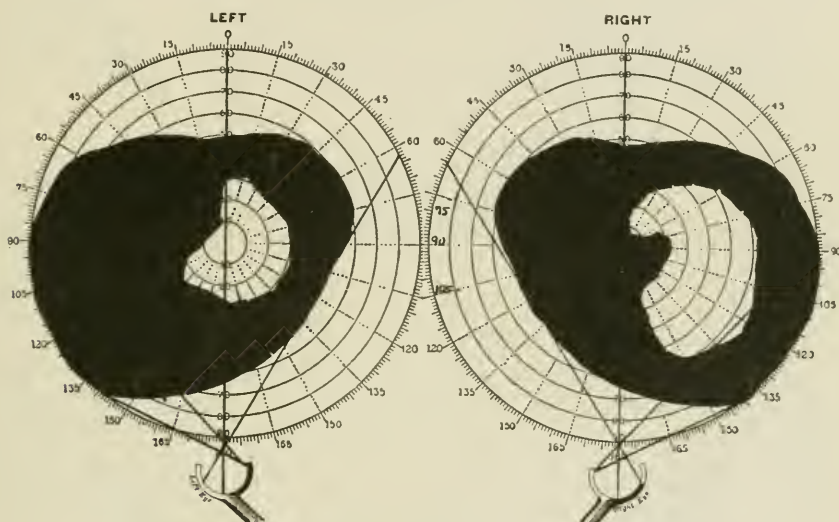


Fig. 4.—Visual fields from a case reported by Cushing.

De Schweinitz and Holloway,⁷ in 1912, were the first to point out (in English) the rôle that these lesions might play in the production of some of the visual field defects seen in pituitary cases. These authors include in this paper a comprehensive review of the literature.

Cushing,⁸ in his monograph on the pituitary and in six later papers⁹ on visual field disturbances in cases of brain tumor, does not emphasize the defects caused by vascular lesions.

The visual fields in one of his cases showed an incomplete left lateral homonymous hemianopia, with marked lower nasal defect on the right side. There is a large cecocentral scotoma passing upward

7. De Schweinitz and Holloway: A Clinical Communication on Certain Visual-Field Defects in Hypophysis Disease, with Special Reference to Scotomas, *J. A. M. A.* **59**:1041 (Sept. 21) 1912.

8. Cushing, Harvey: *The Pituitary Body and Its Disorders*, Philadelphia, J. B. Lippincott Co.

9. Cushing, Harvey: *Brain* **44**:241, 1922. References to all papers are given.

from the outer lower nasal quadrant (Fig. 4). The notching by the artery shown in the drawing seems to impinge at a point where the very tracts that supply this area of the field should pass, and is possibly the cause of the defect in that portion of the field.

Hirsch¹⁰ in a recent survey of the literature, reports the ocular findings in forty-five cases, and contrasts them with those of Uhthoff,¹ whose series contained 148 cases. He reports a large hypophyseal tumor that flattened out the chiasm and pressed it against the anterior cerebral artery, which was found to have deeply notched it transversely. A sketch from his drawing is given in Figure 5.

Walker and Cushing,¹¹ in 1918, reviewed 271 cases of their series, in 148 of which visual field deformities were present. It is of interest to compare the findings of Uhthoff, Cushing and Hirsch, as to visual disturbances.

VISUAL DISTURBANCES WITH PITUITARY TUMORS

Author	No. of Cases	Bitemporal Hemianopia	Homonymous Hemianopia
Uhthoff	148	37.0%	2.8%
Cushing	148	31.7%	14.8%
Hirsch	45	77.0%	0.7%

REPORT OF A CASE

History.—J. R., a white man, aged 31, was admitted Dec. 4, 1921, to the service of Dr. Frazier at the University Hospital, complaining of headache, vomiting and failing vision. He was well until July, 1921, when he began to have severe headaches, paroxysmal in type, both frontal and occipital. In October, his vision began to fail, and he found that he could not read small print. Soon after this he began to vomit, usually in the mornings, but this was not projectile in character. He noted occasional diplopia, but has had no convulsions or ataxia.

Examination.—Physical examination revealed a rather heavy and obese adult in a semistuporous condition, who complained of severe headache. There was no weakness of any cranial nerve. The eyegrounds showed 4 diopters of swelling in the disk on each side. The visual fields were slightly contracted, especially along the temporal margins of the upper portions (Fig. 6). The reflexes of the right upper extremity were increased, and there was marked increase of the reflexes in the lower limbs on both sides. Bilateral abortive ankle clonus was present, but there was no Babinski sign. The spinal fluid pressure was increased to 18 mm. of mercury. The blood and spinal fluid Wassermann tests were negative. The roentgen ray revealed a "top normal" sella (Pancoast). The tentative diagnosis was: increased intracranial pressure, probably from a tumor in the region of the sella. Visual hallucinations became marked.

Operation and Course.—Five days after admission, the patient's condition was such that it was necessary to perform subtemporal decompression. This resulted in improvement.

10. Hirsch, O.: *Ztschr. f. Augenheilk.* **45**, No. 5, 1921.

11. Cushing, Harvey, and Walker, Clifford B.: *Studies of Optic Nerve Atrophy in Association with Chiasmal Lesions*, *Arch. Ophth.* **45**:407 (Sept.) 1916.

December 19, and again January 9, a ventriculogram was made (F. C. G.), and a large defect in filling was shown to be present in the posterior horn of the left ventricle.

January 5, the visual fields (Fig. 7) showed that, in the month since admission, he had developed a left lateral homonymous hemianopia in spite of the decompression. Marked ataxia with increase of reflexes on the left side, numb-

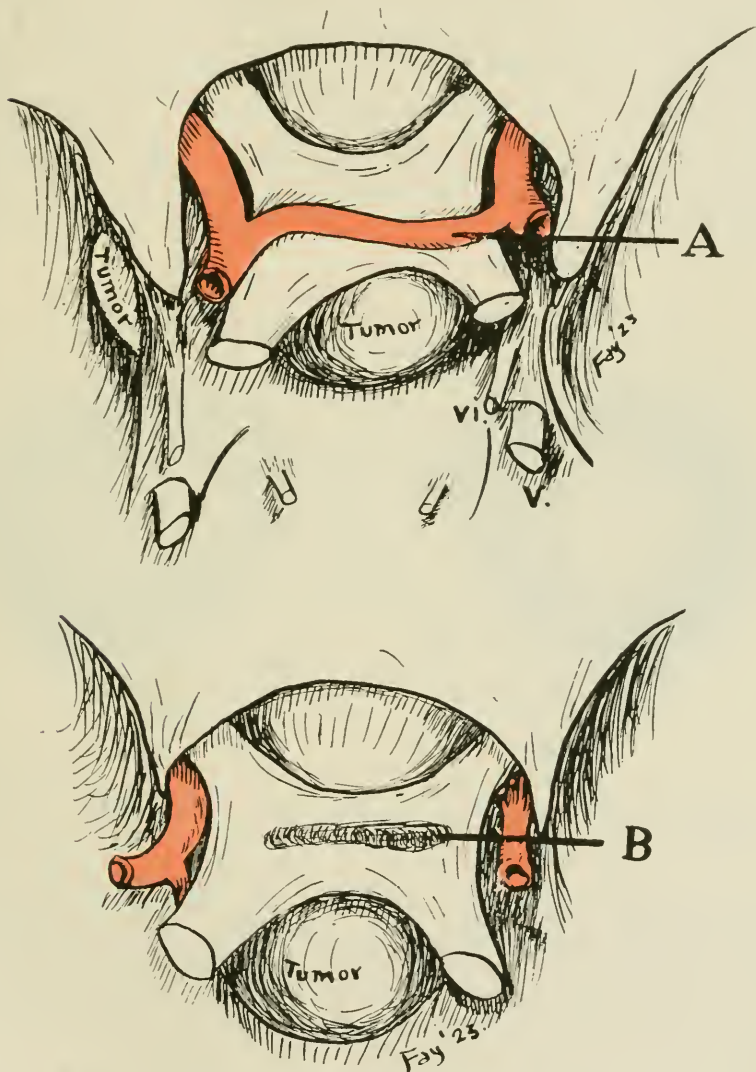


Fig. 5.—Notched chiasm viewed from behind (copied from the report by Hirsch): *A*, anterior communicating artery; *B*, notching of the chiasm by pressure against the anterior communicating artery.

ness, loss of the sense of position, dysmetria, and adiadokokinesis were also present.

Based on these findings, a parieto-occipital exploration on the right side, seemed indicated. The patient was operated on February 3, but no tumor was

disclosed. He made a good recovery, and was up and about the wards for almost a month, but he often dozed, and was aroused with difficulty. He gradually became more stuporous, and finally died on March 23.

Necropsy Examination.—Necropsy revealed a tumor, microscopically an adamantoma, about the size of a walnut, and apparently entirely suprasellar. It was lying in the interpeduncular space just posterior to the chiasm, which had been pushed well forward. The tumor extended into the third ventricle, and posteriorly as far back as the upper margin of the pons (Fig. 7). There was some apparent flattening of the optic tracts and chiasm by pressure from the tumor just below, but on closer inspection, after exposing the sylvian fossa by retraction of the lower borders of the temporal lobes, the optic tracts were found to be notched by the carotid arteries on both sides. On the left side, a deep groove was made on the outer and lower aspect of the tract by the internal carotid artery, 1.2 cm. behind the chiasm. Over the upper and outer portion of the tract, there was a shallow groove caused by pressure

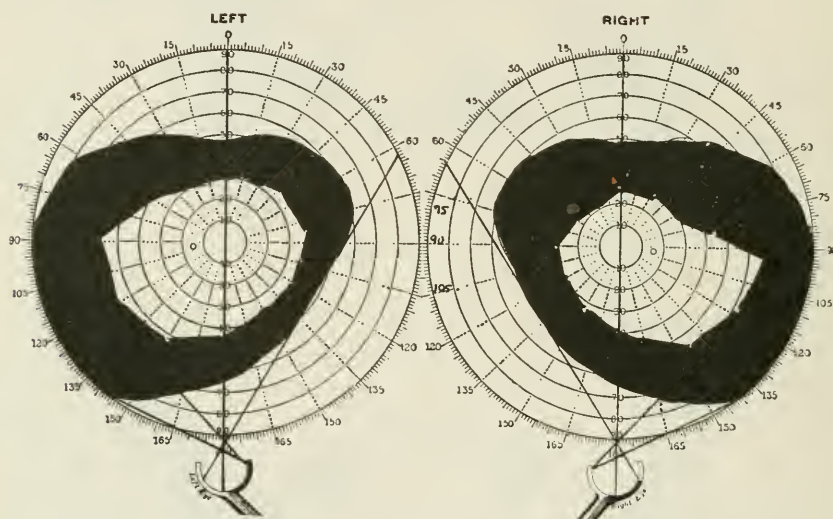


Fig. 6.—Visual fields (with 3 mm. disk) in the case reported, at the time of admission.

against the left anterior cerebral artery as it coursed forward over it. About the center of the chiasm itself, there was a slight dimpling of the structure where the anterior communicating artery exerted pressure. The right optic tract was greatly affected, the fibers having been cut almost two-thirds through by the right internal carotid artery. There was some herniation at the point of decompression, and the cerebral structures had been displaced slightly to the right.

COMMENT

From the visual field charts (Figs. 6 and 7) of this case we may make some interesting speculations.

A general shrinkage of the visual fields was apparent when they were first taken at the time of admission. There was a suggestive upper bitemporal slant which has been so often spoken of by Cushing and is so characteristic of hypophyseal lesions (Fig. 6). The day after these

fields were taken, subtemporal decompression was performed on the right side. Shortly afterward, the ventricles were emptied for a ventriculogram. Twenty-eight days after the first fields were taken the charts showed definite left lateral homonymous hemianopia, which had developed in spite of the decompression (Fig. 7).

Study of the specimen (Figs. 8 and 9) shows that the right optic tracts were so deeply cut by the carotid artery on that side, that only about one third of the tract remained at this point. It may have been that with the relief of pressure on the right, due to the subtemporal decompression, the tumor grew along the lines of least resistance, pressed the structures toward the right and the decompression opening. This would stretch the artery, and since its exit from the cavernous sinus allows but little lateral excursion, we may imagine that the softer

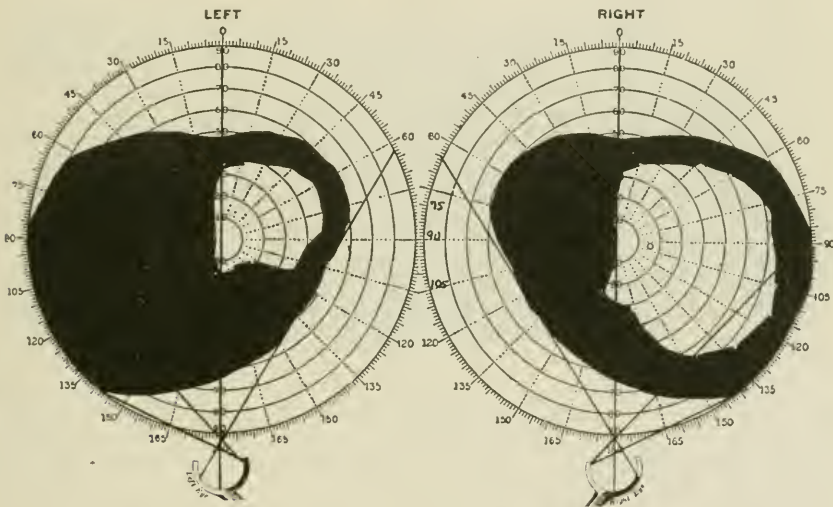


Fig. 7.—Visual fields in the case reported, twenty-eight days after admission and subsequent to ventricular tapings.

structures of the right optic tract lying between the tumor and the pulsating vessel were crushed against it by the enlarging growth, producing an almost complete functional block of that optic tract, and hence lateral homonymous hemianopia on the left side.

The left tract was not only cut by the carotid, but the groove formed was extended forward by the anterior cerebral artery which pressed against the structure as it coursed forward to form the circle. The tract had been caught in the elbow of this branching and the deformity was produced from pressure by the growth from below and medially. Whether the vessel was pulled across the optic tract as a result of the pushing of the structures to the right when tension was relieved by the decompression, with the consequent dragging of the left anterior cerebral artery over the tract on the left, cannot be

determined. Some mechanical factor caused notching of the tract and chiasm by the anterior cerebral artery on the left but not on the right (Fig. 9). It may well be, of course, that the tumor grew more on the left side, and direct pressure was greater here than on the right tract. In either case a left lateral homonymous hemianopia was produced within a month (Fig. 10).

It is of interest to note that the chiasm in this specimen does not ride high on the top of the tumor, but has been pushed forward; and there has been a great deal of pressure exerted on its posterior and lower aspect, with some notching by the tumor. There is no doubt that the fibers have been put on a stretch, and as pointed out by Cushing,¹¹ and reported by de Schweinitz¹² and Holloway, it is here that the papillomacular bundles suffer most.

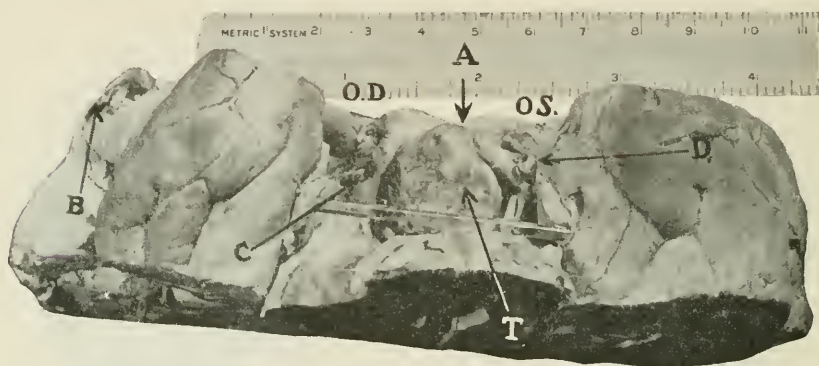


Fig. 8.—Tumor with optic chiasm, tracts and nerves from the case reported: A, the chiasm; B, ventricular hernia at the site of the subtemporal decompression; C, deep notching of the right optic tract; D, the carotid artery and the notched left optic tract; T, the tumor; O D and O S, the right and left optic nerves.

CONCLUSIONS

1. The situation of the optic nerves, chiasm and tracts makes them easily susceptible to injury from surrounding vascular structures in the presence of a rapidly growing tumor in the interpeduncular space.

2. With careful perimetry, defects of the lower nasal quadrants may indicate that lateral pressure is causing physiologic block of the visual pathways; the adjacent and overlying arteries may be responsible for the field defect.

3. Lesions of this character, owing to their concealed position, may easily be overlooked at necropsy.

4. The offending vessels are the internal carotids, the anterior cerebrals and in several cases the anterior communicating artery.

12. De Schweinitz: Concerning the Evolution of Some of the Visual Phenomena of Pituitary Body Disorders, address delivered at opening of Peking Union Medical College, Peking, China, September, 1921, not published.

DISCUSSION

DR. CHARLES H. FRAZIER: This case was confusing because the lesion was of pituitary origin, and yet the patient had a marked papilledema, which we do not usually associate with pituitary lesions. It was a case in which the ventriculogram proved misleading, since the distortion of the ventricle proved to be on the side opposite that of the lesion. There are many cases in which the only, or at least the most important, symptoms are disturbances of the visual fields, and I do not think that we have given sufficient consideration

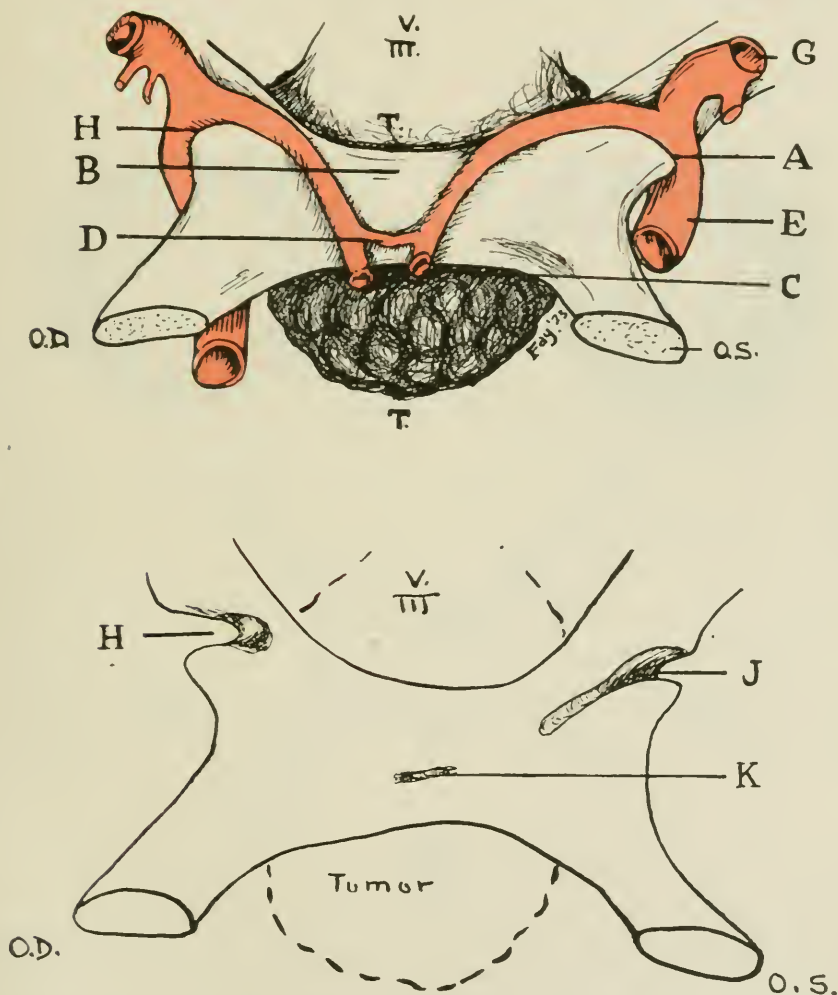


Fig. 9.—Chiasm and optic tracts of the case reported, viewed from in front, illustrating the distortion caused by adjacent arteries. *T*, tumor; *A*, *A*, constriction of the optic tracts by the internal carotid arteries; *B*, the optic chiasm; *C*, anterior cerebral artery; *D*, anterior communicating artery; *E*, left internal carotid artery; *G*, middle cerebral artery; *O D*, and *O S*, right and left optic nerves; *H*, large notch caused by the right internal carotid; *J*, notch caused by the left internal carotid; *K*, depression caused by the anterior communicating artery.

to the part played by the surrounding vessels in the causation of field distortion. If I am correct, the observations which Dr. Fay has made in regard to the effect of the internal carotid artery are original, at least as to the contributions from this country.

DR. FRANCIS X. DERGUM: This communication recalls a case described a number of years ago in which the clinical features were reported by S. Weir Mitchell, and the ophthalmologic findings by Dr. William Thomson, while I described the specimen. The patient presented during life the symptoms of a bitemporal hemianopia, and necropsy revealed an optic chiasm which had

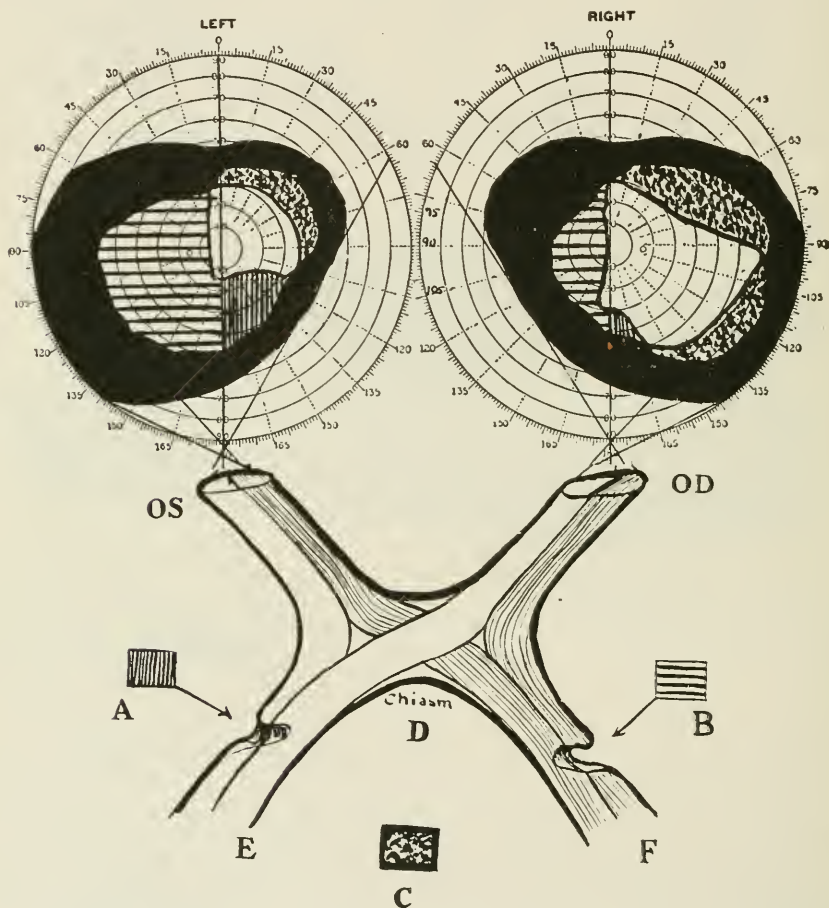


Fig. 10.—Effects of the notching of each optic tract, and of the decompression, on the fields of vision. Interpretation of shading: *A*, defect caused by left internal carotid and anterior cerebral arteries; *B*, defect caused by right internal carotid; *C*, field expansion following decompression; *D*, chiasm; *E* and *F*, left and right optic tracts; *O S* and *O D*, left and right optic nerves.

been divided in the median line by a slowly growing aneurysm of the anterior communicating artery. The divided strands of the chiasm and the optic tracts lay on either side of the aneurysm. The case was an excellent demonstration of the functions of the chiasm. The report was published in the *Journal of Nervous and Mental Diseases*, January, 1889.

EPIDEMIC ENCEPHALITIS

A PATHOLOGIC STUDY OF FIVE CASES INCLUDING
TWO WITH MYOCLONIA

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BOSTON

A systematic and detailed study has been made of five cases of epidemic encephalitis which have come to necropsy at the Boston Psychopathic Hospital. The aim of this work has been predominantly topographic rather than descriptive. In the earlier pathologic studies, attention was naturally focused on the midbrain; later researches have revealed that the changes in the nervous system are more widespread than was at first apparent. Recently these outlying lesions have received more study and the name "neuraxitis" has been proposed by French writers as more truly expressing the conditions found. Two of the cases in this series were of the myoclonic form, on which there are as yet few complete pathologic reports.

The cortical areas examined in each hemisphere were: the first frontal, the precentral, postcentral, temporal, calcarine, and hippocampal. Four or five blocks were made from the corpus striatum of each side, and two each from the thalamus and hypothalamus. Practically the whole of the midbrain, pons and medulla were cut into blocks. Three and frequently more levels of the cord were examined. The cranial nerves, the gasserian and posterior root ganglions, and the peripheral nerves were examined whenever they had been removed. The stains used were cresyl violet, Mallory's phosphotungstic acid hematoxylin and scharlach R. Several attempts were made with Alzheimer's methods for degeneration products and ameboid glia, but they were unsatisfactory as the tissue had been hardened in formaldehyd solution. The trunk organs and the pituitary were also studied in each instance.

REPORT OF CASES

CASE 1.—W. B. A., a man, aged 38, an electrician with a past history that had no bearing on the present illness, developed, March 23, 1922, a bad head cold. Four days later he suddenly became confused, spoke of violet rays coming through his body and feared that they would kill him. March 29, he became restless and had to be held in bed. He would put his hand to the back of his neck as if in pain. At no time was there any complaint or indication of double vision. March 30, he was admitted to the Psychopathic Hospital. His temperature then was 101.4; pulse, 116. He did not cooperate in the examination. There was no obvious defect in the cranial nerves; the pupils reacted well to light; gait and station indicated general weakness; there was slight cyanosis of the hands; the knee and ankle jerks were active and equal, as were also the triceps and abdominal reflexes. Examination of the chest and abdomen revealed no special abnormality.

Course.—During his stay in the hospital his behavior was in general of the katatonic type. He occasionally cooperated and answered questions, but for the most part he was uncooperative or negativistic; at times he was actually resistive. He frequently assumed rigid attitudes. He admitted that he experienced auditory and visual hallucinations, but he did not react to them. Underlying this behavior there was, however, a severe general intoxication. He became rapidly weaker with marked muscular relaxation, slight cyanosis and an increasingly weak and rapid heart. April 3, there appeared to be a divergent strabismus. He died April 5, the duration of the illness being twelve days.

CASE 2.—E. P., a young French woman, aged 26, a teacher in a private school, with a past history that is not of interest in the present connection, was vaccinated Nov. 23, 1920, and had a good reaction five days later. November 26, "everything seemed to be trembling" when she awoke and she saw double. This symptom lasted for six days and was most noticeable in the morning. The next day when she tried to get up she found she could not walk and she also had difficulty in speaking. The following morning she could not use her arms and she had trouble in swallowing. There was no pain or discomfort and no unusual sleepiness. The physician's examination at this time showed that pulse, temperature and respiration were normal. The uvula was edematous and covered with a profuse mucous exudate (negative for diphtheria bacilli). The pupils were equal and somewhat dilated; there was slight twitching of the eyelids. The superficial and deep reflexes were normal; muscular coordination was good and there was no paralysis. The tentative diagnosis was hysteria. She continued in about the same condition until her admission to the Psychopathic Hospital on December 12.

Examination.—On admission she felt very weak and uncomfortable, but had no actual pain. She was mentally clear and alert, quiet and cooperative. The neurologic examination at this time showed: pupils widely dilated, the right slightly the larger; both reacted in a limited degree to light and better on accommodation; partial bilateral ptosis; transient external strabismus of the left eye; facial movements were good with the exception of the orbicularis oris; marked difficulty in speaking and in protruding the tongue, which was, however, in the midline and showed no tremor; much difficulty in swallowing; mastication normal; hypersecretion of mucus in the throat. The patient could move the arms and legs but said they felt heavy; power was fairly good against resistance; finer movements of the fingers were poorly carried out. Tendon reflexes: biceps sluggish; triceps not obtained; patellar and Achilles normal. There was no Babinski sign. Some difficulty existed in urination. The chest and abdomen were negative.

Course.—The patient seemed to improve during her stay in the hospital. She was stronger and had less difficulty in talking and swallowing. She died very suddenly, December 19, while being given a bed bath. The duration of the illness was twenty-three days.

CASE 3.—E. M. D., a girl aged 16, is especially interesting both from a psychiatric standpoint and because there is the possibility of two cases of epidemic encephalitis in the same family. The history was essentially negative up to the time of her last illness. A few months previously the patient's twin sister (L. D.) had been the victim of an assault and since had been depressed and worried over the matter. Early in March, 1922, she (L. D.) was taken with "grippe." Two days after the onset she became confused and violent. Attacks of great excitement alternated, at first, with quiet and a

return to normal mentality, later with stupor. She continued in the same condition until her death at home, April 16. The diagnosis made was dementia praecox. Our patient (E. M. D.) was deeply attached to her sister and had been much impressed by the assault. She was with her sister constantly during the first week of the latter's illness. March 20, she was seized with a convulsion and following this she was restless, sleepless, refused to eat or talk, and became violent when any effort was made to control her. Later she was in a stupor most of the time. She was admitted to the Psychopathic Hospital April 18.

Examination.—On admission the patient was restless and did not cooperate. At times she showed some flexibility and was negativistic. Physical examination revealed: much emaciation; marked diminution of tactile sensibility; pupils dilated but reacting to light. There was an Oppenheim sign on the left. Other neurologic tests were negative. The general examination was negative, except for a very rapid heart rate (132). The spinal fluid showed no globulin, normal albumin and a negative colloidal gold test. The temperature was 100.8 on admission, but was subnormal for two days before death. The patient vomited her tube feedings, became weaker, cyanotic, and died April 20, after an illness of one month. This case might easily have been interpreted from a psychogenic standpoint, the mental symptoms representing a hysterical repetition of her sister's illness but a consideration of all the factors makes the organic basis of the condition clear.

Cases 4 and 5 were examples of the myoclonic form of the disease.

CASE 4.—J. L., a man, aged 38, a clerk with a past history that is unimportant, noticed in the latter part of December, 1920, numbness of the hands and stiffness of the fingers. About Jan. 4, 1921, he stopped work on account of headache; January 18, this was severe enough to cause him to go to bed. He was very irritable, talkative and restless at this time. The numbness in his hands persisted and about January 14, he had pains in his feet and legs. He was unable to sleep on account of the pain and headache. January 20 his temperature was 101. Twitchings of the trunk and extremities were first noted by the family on January 22. He was then sent to a general hospital, where he soon became confused and hallucinated. He was admitted to the Boston Psychopathic Hospital January 25. Here he was restless, confused and disoriented, pulled at the sheets, thinking it was clothing to put on, talked to his children at home, etc.

Examination.—Physical examination disclosed: the pupils reacted sluggishly to light; nystagmus of both eyes, increased on lateral or upward movement; weakness of the levator palpebrae and an inability to close the eyes completely; the deep reflexes were slightly increased, especially the knee jerks; there was no ankle clonus. The abdominal muscles showed shock-like contractions, occurring from twenty to thirty times a minute. These contractions extended occasionally into the muscles of the thighs, causing them to flex on the abdomen. The movements of the thighs and abdominal muscles were simultaneous. They were more marked on the right side and were accompanied by severe pain. The abdomen was slightly tender on pressure. On the two days before death the abdominal contractions decreased to ten or twelve a minute. There were also occasional twitchings of the arms and marked coarse tremors of the forearms and outstretched fingers, the latter increasing on voluntary movement.

The temperature gradually rose from 101.4 on admission to 104 on the day of death. The patient passed into coma and died February 1, after an illness of four weeks.

CASE 5.—A. H., a man, aged 31, a factory operative, with a past history that is unimportant, for two months complained that he felt run-down and could not work as well as formerly. About three weeks before admission to the Boston Psychopathic Hospital he was said to have had influenza, although he was in bed only one day. A week later, March 26, 1922, after doing some work around the yard of his home, he complained of pains in his arms. These grew so severe that they prevented sleep. He became delirious at night and twitchings in the arms were noticed. After a few days he was constantly delirious and complained of headache and pain in the neck and back. He was brought to the hospital April 5.

Examination.—Physical examination showed the pupils almost rigid to light but reacting on accommodation. There was a lateral nystagmus. The deep reflexes were present in the upper extremities; the right knee jerk was absent, the left diminished; the ankle jerks were normal. There was a tendency to a Babinski sign on the right. There were clonic contractions of the muscles of the upper part of the abdomen, occurring about twenty-five times a minute. These occurred in cycles of three to seven strong contractions, then a few weaker ones, followed by an interval of rest; then a repetition. The patient seemed to hold the muscles tense and in this way he could control the movements to a slight extent. There was no abdominal tenderness. There were also twitchings of muscle groups in the right arm and forearm and at times irregular jerkings of the legs.

The temperature varied from 100 to 101 for five days after admission. April 11, signs of pneumonia were apparent and the patient died April 12, after an illness of seventeen days.

HISTOLOGIC FINDINGS

Interest in the cerebral cortex in lethargic encephalitis has been subsidiary to that in the midbrain and lower centers, and consequently much less attention has been devoted to it. It has frequently been pronounced negative after examination of a few sections. Some writers have found neuronophagia, hemorrhages and slight perivascular exudate. Others, who have been interested primarily in the finer histology of the disease, have described the lesions without particular reference to their situation, although they mention certain of them as occurring in the cerebral cortex. Hassin¹ has emphasized the difference between toxic and infectious encephalitis, using lethargic encephalitis as an example of the latter type; he contrasts the productive lesions of the toxic with the infiltrative lesions of the infectious type of encephalitis. Buzzard² and Watson³ have reported cases of lethargic encephalitis in which the maximum of the disease was found in the cortex, but their cases differed much from the ordinary form of the disease clinically,

1. Hassin, G. B.: The Contrast Between the Brain Lesions Produced by Lead and Other Inorganic Poisons and Those Caused by Epidemic Encephalitis, *Arch. Neurol. & Psychiat.* **6**:268 (Sept.) 1921.

2. Buzzard, E. F.: Lethargic Encephalitis, *Lancet*, **2**:835 (Dec. 21) 1918.

3. Watson, G. A.: Encephalitis Lethargica Involving Chiefly the Cerebral Cortex, *J. Neurol. & Psychopath.* **1**:34 (May) 1920.

by showing paralyses and aphasia, and pathologically, by having large areas of softening and hemorrhage; further, no mention is made of the lesions in the midbrain.

As no special study seems to have been made of the distribution of the cortical lesions in epidemic encephalitis, our attention was directed particularly to this point, to see whether, like the changes in general paralysis, they predominated in any special areas.

Definite changes were found in the cortex in all the brains. They were of the same general nature in all, though they differed in intensity and relative prominence in the different cases. Cell changes were slight in all except Case 1. They consisted in mild chromatolysis and occasional shrunken and diffusely staining cells with eccentric nuclei. The affected cells were scattered among normal ones in all layers. In Case 1, which was of the shortest duration (twelve days), the changes were marked and disintegrating cells were numerous.

Increased satellitosis was also present in all cases, though it varied considerably from case to case and section to section. It was most abundant, as is usual in the deeper layers of the cortex. Neuronophagia was evident only in Case 1, in which it was quite prominent. Neuroglia cells having large, light-staining nuclei and visible cytoplasm were numerous in Cases 1 and 5, especially around the nerve cells; they probably represented transitional stages to ameboid glia.

Perivascular infiltration was slight, but was found in all cases, though not in all areas. Usually one or two vessels to a section were surrounded by lymphocytes. Plasma cells were sometimes found in the tissue. Congestion of the cortical vessels was seen in all sections, and occasional hemorrhages into the perivascular spaces were present.

Considerable accumulations of degeneration products, presumably lipid, occurred around the vessels in all cases. These stained green with cresyl violet and brownish with phosphotungstic acid hematoxylin, and were present chiefly around the veins in the white matter. The fat-content of the cortex was very moderate in all except Cases 2 and 3, in which there was a heavy deposit around the vessels and in the nerve cells in all regions. It is interesting to note in this connection that the mentality of the second patient was practically normal.

In regard to the topography of the above described lesions, no special distribution could be made out. They were scattered irregularly through the various cortical areas, and the occipital region was as often affected as the frontal or central. The congestion and the lipid deposits were especially diffuse in their distribution.

The changes in the corpus striatum were of the same general nature—chromatolysis, satellitosis, slight perivascular exudate, and lipid pigment. There was also some diffuse infiltration of the tissue and clumps of glia nuclei. The lesions were slight in all cases except Case 1, in which, as has been said, the changes in the cortex were more marked and more acute than in the other brains.

In Case 1 the putamen showed a rather unusual degeneration product, which appeared as homogeneous globules of various sizes, that stained an intense uniform dark blue with cresyl violet and hematoxylin. They were found chiefly among the fiber bundles as scattered clusters or as beading along the vessels. They were present nowhere else in the nervous system nor in any other case. Similar bodies have been described in the basal ganglions in epi-

demic encephalitis by Calhoun;⁴ also by Faworsky⁵ among the degeneration products in the cord in tabes. Fulci⁶ has described, in a case of anthrax encephalitis, what seems to be a similar material, but in his case it occurred in large masses that filled the perivascular spaces. The nature of this substance is unknown, but it apparently belongs among the basophilic degeneration products of Alzheimer.

The thalamus and hypothalamus presented the same lesions as the corpus striatum. In general they were moderate in degree, and in Case 2 they were almost lacking. Both thalamus and hypothalamus were equally affected.

The midbrain was in all instances the site of the usual intense lesions. It may be mentioned that the red nuclei, the corpora quadrigemina and the geniculate bodies shared in the general marked involvement.

The changes in the cerebellum were slight and consisted of foci of exudate in the pia, occasional cloudy swelling of the Purkinje cells, small collections of lymphocytes in the white matter, and very rarely a little perivascular exudate.

The lesions in the brain stem and cord are considered in the discussion of the myoclonic cases, as it was in these regions that differences were found between the myoclonic cases and the others.

The optic nerves were examined in all cases. In Case 1 there was fresh hemorrhage into the pial sheath, and in Case 5 hyaloid droplets were present. The others were normal. There were small collections of lymphocytes in the olfactory tract in two instances.

Exudate was frequently present among the roots of the other cranial nerves at their points of origin, but it did not extend far outward. Burrows⁷ has particularly emphasized the involvement of the cranial nerves, but the changes found in our cases were not as great as he reports.

Nerve bundles from the cauda equina were normal in all cases except Case 5, in which there was slight focal perivascular infiltration.

The gasserian ganglions showed changes in Cases 1, 3 and 5. The cells stained diffusely or were shrunken and pyknotic; the vessels were congested and there was considerable diffuse lymphocytic infiltration, which in one instance extended out among the nerve bundles. Neuronophagia and hemorrhage were absent. The writer has frequently found infiltration of the gasserian ganglions in various kinds of cases, not only when there was an inflammatory lesion at the base, as meningitis, but also in syphilis of the nervous system, and in noninflammatory conditions such as dementia praecox, alcoholic dementia, etc.

Specimens of the posterior root ganglions were examined in three cases, including one of the myoclonic. They were all normal.

No changes were observed in the ependyma of any of the ventricles or of the aqueduct.

The choroid plexus was examined in three cases. Two presented no abnormality but the plexus of the fourth ventricle of Case 5 showed a large

4. Calhoun, H.: Histopathology of the Brain and Spinal Cord in a Case Presenting a Post-Influenzal Lethargic Encephalitic Syndrome, *Arch. Neurol. & Psychiat.* **3**:1 (Jan.) 1920.

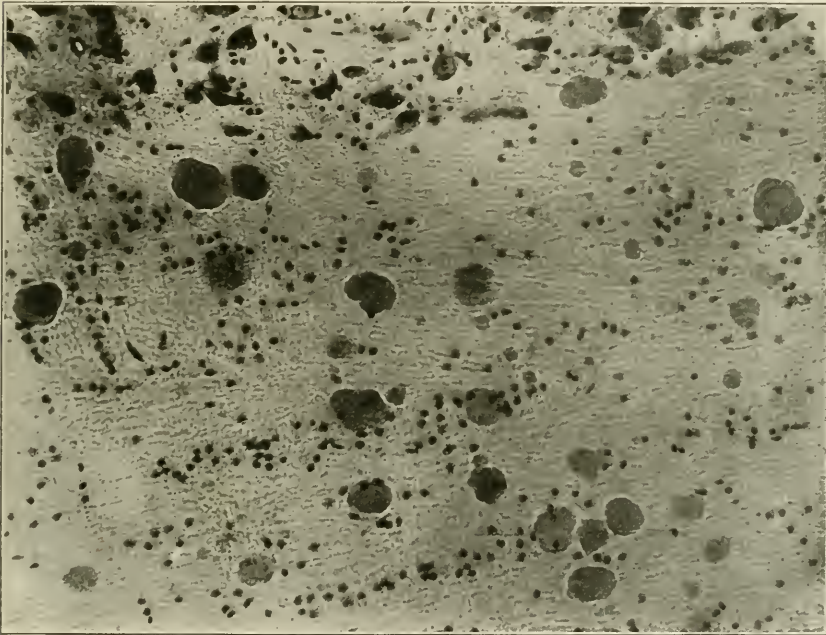
5. Faworsky, A.: *Histol. u. histopathol. Arb. über die Grosshirnrinde*, Nissl and Alzheimer **6**:74, Part 1, 1913.

6. Fulci, F.: *Histol. u. histopathol. Arb. über die Grosshirnrinde*, Nissl and Alzheimer **6**:160, Part 2, 1913.

7. Burrows, M. T.: Neuritis of the Cranial Nerves in Lethargic Encephalitis, etc., *Arch. Int. Med.* **26**:477 (Oct.) 1920.

vessel containing a white thrombus. The surrounding plexus contained considerable fresh hemorrhage.

A noteworthy finding was the presence in Case 2 of an enormous amount of a degeneration product resembling colloid. This appeared as sharply outlined refractile droplets of various sizes (shown in the accompanying figure). The larger masses frequently had convoluted edges. They stained bright pink with thionin, extremely pale pink with cresyl violet and phosphotungstic acid hematoxylin and were unstained by alum hematoxylin, eosin and scharlach R. With Best's method for glycogen they took a pale mauve tint. They were most abundant in the midbrain and upper pons, where both gray and white matter were studded with them. They were abundant also throughout the rest of the pons and in the medulla, numerous in the corpus striatum and thalamus, and



Midbrain, (Case 2), showing the large amount of colloid material.

present in small numbers in the various cortical areas. They were also scattered through the white matter of the cervical cord, but not below this level. Degeneration products, similar in a general way to the above, have been described in epidemic encephalitis by Calhoun,⁴ Boyd⁸ and Harris.⁹ Watson³ also has described and pictured, in the neighborhood of vessels, rarefied sieve-like areas which frequently contain an amorphous deposit staining deep mauve with toluidin blue, lighter mauve with Van Gieson's stain and brownish orange with pyronin methyl green. These substances described by the various writers vary somewhat in staining reactions, judging from the accounts given,

8. Boyd, W.: *Acute Epidemic Encephalitis*, New York: Paul B. Hoeber, 1921, p. 189.

9. Harris, A.: *A Case of Epidemic Encephalitis*, *Am. J. Psychiat.* **1**:679 (April) 1922.

but they all seem to belong to the ill-defined group of so-called "colloid" of the central nervous system. Boyd surmises that they are derived from neuroglial degeneration.

The changes described above in the cortex, basal ganglions and midbrain were common to all the cases. In the study of the brain stem and cord it was found that both the myoclonic cases presented a similar picture with minor variations and differed from the others in two respects; first, in the greater intensity of the process in the pons and upper medulla; and second, in the presence of lesions in the cord.

In regard to the pons and medulla, the myoclonic cases exceeded the others in the degree and extent of the exudate rather than in the nerve cell lesions. The latter were prominent in all the cases and consisted in scattered degenerating cells, numerous cells showing chromatolysis, and considerable satellitosis. The affected cells were most numerous in the dorsal portion, where they were scattered through all nuclei. They were not confined to the dorsal side, however, but were present to some extent also in the pontine and olivary nuclei. On the whole, it is impossible to say that one nucleus in the dorsal region was more involved than another, except that the locus ceruleus always showed marked lesions, and abnormal cells were always numerous near the raphe and in the formatio reticularis. Perivascular exudate was abundant and widespread throughout the pons and medulla in the myoclonic cases. In Case 4 it was heavier in the upper pons than in the midbrain. There was also a diffuse increase of nuclei throughout the tissue and proliferation of the vascular endothelium. The pia was infiltrated throughout the length of the medulla. The intensity of the lesions, both cellular and infiltrative, diminished as the lower levels of the medulla were reached. In the non-myoclonic cases exudate was scanty and was absent in many sections.

Involvement of the cord was evidenced macroscopically in Case 5 by fresh adhesions between the dura and pia in the cervical region and by the presence of bloody fluid in the thecal sac. Both myoclonic cases showed marked perivascular exudate in the lower cervical and a lesser amount in the lumbar cord. In the cervical region the exudate appeared both in the gray matter of the anterior and posterior horns and in the white matter; in the lumbar cord only in the anterior horns. Exudate was limited to the cervical and lumbar enlargements but the nerve cell changes were more widely distributed. They consisted chiefly in cells showing the axonal reaction, which were found in the cervical and lumbar and also in the dorsal regions. The dorsal cord was the least involved in both instances, showing nothing beyond a few cells giving the axonal reaction. This is particularly noteworthy, as the muscles most concerned in the myoclonic movements were the recti abdominis, which are innervated from the fifth to the twelfth dorsal segments.

The pia in the cervical and lumbar regions was mildly infiltrated. Both anterior and posterior roots, as they appeared in sections, were normal.

The non-myoclonic cases showed no exudate. The gray matter was congested in all cases and in Case 3 there were two extensive areas of hemorrhage into the perivascular spaces of the posterior horn of the fourth cervical segment. Minor cell changes were present in the cervical region of Case 1.

In summary then, the distribution of the lesions in the myoclonic cases was as follows: diffuse and mild in the cortex, corpus striatum and thalamus; of the usual intensity in the midbrain; very marked also in the pons; decreasing

gradually throughout the medulla; marked again in the cervical region of the cord; mild or absent in the dorsal region; and slight in the lumbar region of the cord.

In some of the reported necropsies on cases of epidemic encephalitis showing movements of the myoclonic type, the lesions have been described as unusually severe in the pons and medulla; and when the cord has been examined, it has generally been found involved.

In Winkelman and Weisenburg's¹⁰ case there were twitchings all over the body, and pains which were sharply limited to the head, upper limbs, chest, and abdomen to the level of the umbilicus. "The pathologic findings were those which are usually present, but careful study of the cord demonstrated that there was a lessening of the pathologic process as the lower levels of the cord were reached, and that the cord below the lower thoracic level was altogether normal."

In Hassin's¹¹ case there was enormous infiltration in the pons, midbrain and locus niger, both perivascular and invading the parenchyma. The cord was not removed.

Gerlach¹² reports two cases, both of the lethargic type, that showed muscular twitchings; in the first patient these involved the entire body; in the second, the abdominal muscles and one hand. In both cases the lower dorsal and upper lumbar regions were involved, the other parts of the cord only slightly. There were marked changes in the medulla.

Häuptli¹³ gives short accounts of three cases in which the cord showed marked infiltration. In the first there was pain in the abdomen and left arm, twitchings in the latter, hyperalgesia of the left leg, increased knee jerks, and ankle clonus. The cervical cord was diffusely infiltrated. In the dorsal cord exudate was less abundant and was focal. The second case showed, at the onset, pain in the right shoulder, abdominal muscles and right leg, and later, clonic contractions in these groups, also hiccough. The maximum of the lesion was not in the midbrain but in the medulla, and it continued with special severity throughout the entire cord. In the third case there were myoclonic twitchings in the arms. The lesions were less intense in the midbrain and medulla than in the cerebellum and cervical cord.

10. Winkelman and Weisenburg: A Case of Myoclonic Lethargic Encephalitis Presenting Unusual Pathologic Manifestations, *Arch. Neurol. & Psychiat.* **5**:224 (Feb.) 1921.

11. Hassin, G. B.: Two Cases of Atypical Epidemic Encephalitis, *J. Nerv. & Ment. Dis.* **53**:217 (March) 1921.

12. Gerlach, W.: Ueber Rückenmarksveränderung bei Encephalitis lethargica, *Berlin klin. Wchnschr.* **57**:585 (June 21) 1920.

13. Häuptli, O.: Zur Histologie der Poliomyelitis acuta und der Encephalitis epidemica, *Deutsch. Zeitschr. f. Nervenhe.* **71**:1 (May) 1920.

Ducamp and Carrieu¹⁴ report a case of epidemic hiccough with myoclonia. The lesions were especially localized in the pons, medulla and cervical cord. The lesions in the peduncles were less intense than usual. In the medulla, degeneration was found principally in the vago-spinal nucleus. The dorsal and lumbar cord were only slightly affected.

Roubier¹⁵ reports three myoclonic cases in which the lesions were most marked in the pons and locus niger.

Valente and David,¹⁶ in three necropsies on the pure myoclonic form of Sicard, found diffuse, very grave lesions of the cord, striking, with evident election, the segments which corresponded to the most marked pains and myoclonic movements. In one case the authors found no lesions above the medulla; in the two others there were pontine and midbrain lesions, with normal basal ganglions and cortex. The lesion was a total acute poliomyelitis, striking the gray matter in its entirety.

In Bostroem's¹⁷ and Bradley's¹⁸ cases the findings were different from those given above. In Bostroem's two cases the most affected areas were the floor of the fourth ventricle, the brachium conjunctivum and the lenticular nucleus, particularly the median part of the globus pallidus. No mention is made of the cord. In Bradley's case the lesions were in the midbrain and pons. The medulla showed little abnormality and the cord none.

Our own results are indecisive as far as locating any particular lesion responsible for the myoclonia is concerned, and most probably nothing so definite and elementary is to be expected. Although there is a tendency for myoclonic cases to show lesions in the cord, it does not seem as if these could be directly connected with the movements, for at least one necropsy has been reported in which the cord was negative; on the other hand, there have been cases described in which the cord was much involved but no myoclonia was present. Moreover, the distribution of the cord lesions, in our cases at least, is not what one would expect, for although the recti abdominis were the muscles most affected, the dorsal cord, from which they are innervated, is the least involved portion. The variation of the pathologic findings in the reported cases shows the complex and unsettled state of the question.

14. Ducamp and Carrieu: Hoquet épidémique avec mouvements myocloniques généralisés, *Bull. Acad. d. méd. de Paris* **86**:249, 1921.

15. Roubier: *Lyons Méd.* **129**:452, 1920.

16. Valente and David: Sur la poliomyélite myoclonique, *Compt. rend. Soc. biol. de Paris* **83**:1390 (Oct. 25) 1920.

17. Bostroem, A.: Ungewöhnliche Formen der epidemischen Enzephalitis unter besonderer Berücksichtigung hyperkinetischer Erscheinungen, *Deutsch. Zeitschr. f. Nerven.* **68**: and **69**:64 (Jan.) 1921.

18. Bradley, E.: A Case of Myoclonic Epidemic Encephalitis, *Brit. M. J.* **1**:891 (June 18) 1921.

The prevailing opinion from clinical evidence in regard to the site of the lesion responsible for myoclonic movements is that it is probably an irritative lesion of the lower motor neurone. The lancinating pains which are characteristic of the myoclonic form have suggested a participation of the posterior root ganglions in the pathologic process and also that they may be the point of entrance of the virus. The former idea has not been borne out by the one of our myoclonic cases in which the ganglions were examined. Both Mingazzini²⁰ and Walshe²¹ consider the myoclonia due to infiltration of the anterior roots. As has been said, the anterior roots in all our cases were normal.

The findings in the organs of the trunk were as follows: all the organs showed congestion, which was most striking in the spleen. In Cases 2, 3 and 4 the lungs contained areas of fresh hemorrhage, and in Cases 3 and 4 there was an early bronchopneumonia. The intestinal tract, in Cases 1 and 4, was the site of definite lesions. In the former the intestine, especially the ileum, was injected macroscopically and in the lower ileum the mucosa was thickened and covered with fluid blood. The mucosa of the cecum was thickened and presented numerous ulcers, which had red edges and yellow bases, beneath which was hemorrhage showing through on the peritoneal coat. The cecal lymph nodes were enlarged and purple. Microscopically the thickened areas showed a superficial necrosis, and beneath the surface were many large mononuclear phagocytes. The lymphoid tissue was increased in amount and active. The vessels were injected and those in the muscular coat were lightly infiltrated with lymphocytes. In Case 4 the gastric mucosa showed petechial hemorrhages and the upper part of the duodenum and lower portion of the ileum were much injected. The congestion and petechial hemorrhages are similar to those frequently found in other organs in epidemic encephalitis, but the changes in Case 1 are unusual. The significance of this lesion and whether it has any relation whatever to the disease is problematic. There is no mention of intestinal symptoms at any period of the disease. Lymphoid hyperplasia, either in the intestine or other organs, is not a part of the pathology of epidemic encephalitis as it is of poliomyelitis, nor are intestinal symptoms frequent, although Dana²² mentions their occurrence at the

20. Mingazzini, G.: Klinischer und anatomisch-pathologischer Beitrag zum Studium der Encephalitis epidemica, *Zeitschr. f. Neurol. u. Psychiat.* **63**:199, 1921.

21. Walshe, F. M. R.: On the Symptom-Complexes of Lethargic Encephalitis with Special Reference to Involuntary Muscular Contractions, *Brain* **43**:197 (Nov.) 1920.

22. Dana, C. L.: *Acute Epidemic Encephalitis*, New York: Paul B. Hoeber, 1921, p. 133.

beginning and Barker²³ during the course of the disease. There seems to be nothing in the literature on lesions of the intestinal tract in epidemic encephalitis aside from an occasional description of congestion found at necropsy.

The pituitary gland was normal in all cases except for congestion. In three instances there seemed to be an unusually large proportion of basophil cells in the pars anterior in sections taken directly through the center of the gland, but the distribution of basophils and acidophils is normally so irregular that it is difficult to judge of their proportions.

SUMMARY

Systematic examination of various parts of the central nervous system in epidemic encephalitis has shown that although centered in the midbrain, the disease process affects all parts of the cerebrospinal axis. Practically no region is entirely normal. Except in the midbrain the lesions are scattered and focal, the affected cells and vessels being found among others which appear quite normal.

In the cerebral cortex the lesions are irregularly and diffusely distributed and no one region is more affected than another. The same cortical changes are found in patients who have shown very different mental symptoms.

The myoclonic cases were distinguished from the others by the greater severity of the process in the pons and medulla and by involvement of the cord. The parts affected were the cervical and to a lesser extent the lumbar regions.²⁴

Various degeneration products in the central nervous system and the presence of intestinal lesions are described.

Boston Psychopathic Hospital.

23. Barker, L. F.: *Acute Epidemic Encephalitis*, New York: Paul B. Hoeber, 1921, p. 133.

24. Since this article was written another case of epidemic encephalitis with myoclonia of the abdominal muscles has come to necropsy at the Boston Psychopathic Hospital. The sections show an enormous infiltration in the midbrain and the dorsal portions of the pons and medulla. In the cervical and thoracic regions of the cord there is also a heavy perivascular infiltration in the posterior horns, and around occasional vessels in the white matter. The lumbar cord is not affected.

STABILIZING BRAIN TISSUE DURING FIXATION

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In the fixation of tissue it is the aim to hold the structures as nearly as possible in the form they had during life, or at the moment when the fixing reagent acted. Exceptionally, an artefact may have a differentiating value; with this we are not concerned here. But when conditions exist which can be recognized as artefact, it is desirable to eliminate them, or to evaluate them.

At present 10 per cent. liquor formaldehydi, U. S. P., is the most extensively used fixative for whole brains. In dealing with unknown brain tissue so fixed, conclusions regarding deviations from the normal picture are based on our experience with formaldehyd fixed tissue. Certain conditions due to fixation rather than to antemortem influences are eliminated; for example, certain clouding of the pia-arachnoid is expected to appear after exposure to formaldehyd. In finer histologic work, detectable change in structure, due to fixation, is apt to vary in intensity and must be considered objectionable, and should be eliminated if possible. Such changes may obscure or be confused with actual antemortem changes.

Two conditions found after the fixation of brains in 10 per cent. liquor formaldehydi, U. S. P., prompted a study of some of the changes attending such fixation, and a search for methods of preventing those that were objectionable. Two conditions that we considered undesirable, are apt to occur in brain tissue whether pathologic changes are present or not. They were especially brought to our attention since they appeared in tissues intended for use as normal controls.

The first of these two changes was the swelling and change of weight that took place after the time of necropsy and during fixation. It is fairly well known that a brain immersed in 10 per cent. liquor formaldehydi at necropsy gains considerably in weight for a while, increasing usually 100 or more grams. It then falls again, but usually does not return to its original weight. If after fixation such a brain be transferred to a solution of 10 per cent. liquor formaldehydi in physiologic sodium chlorid solution, it falls below its original weight. The following observation is an example of such a change:

A brain at necropsy (Nov. 13, 1920) weighed 1,698 gm. and was placed in 5 per cent. formaldehyd solution. Two weeks later, Nov. 29, 1920, the brain

weighed 1.805 gm., a gain of 107 gm., and was then transferred to 4 per cent. formaldehyd in physiologic sodium chlorid solution. Subsequent weights in grams were:

Dec. 13, 1920.....	1,745
Dec. 27, 1920.....	1,705
Jan. 19, 1921.....	1,675
April 15, 1921.....	1,651
May 18, 1921.....	1,635
Aug. 3, 1921.....	1,620

We are faced with the problem of finding a method of fixing tissues that would reduce these volume alterations.

The second change consisted of the so-called shrinkage alteration of the nerve cells of the second and part of the third layer, or of those even deeper. While not a constant change, it was so frequent as to excite doubt as to its pathologic significance. Aside from its own significance, it is a change that would be apt to obscure a slight, but truly pathologic, alteration in these cells. The claim has been made by Hoch,¹ that such changes sometimes arise from treatment during fixation and other technical procedures. He remarked in 1898 that "we . . . (cannot) . . . more than indicate the difficult problem which arises from the combination of this with other changes." He was able to duplicate these changes by treating fresh rabbit brain tissue with distilled water. Dunlap has called attention to the fact that the cortex deep in the sulci, where protected from rapid fixation, was usually free from this alteration. He suggested that some action on the unprotected surface probably had a bearing on this change. Whether the practice of washing the surface blood from a brain prior to fixation has anything to do with this change is a question. At any rate it was considered desirable to eliminate it if possible.

From our experience with the behavior of brains after treatment with formaldehyd and with formaldehyd and sodium chlorid, we concluded that alteration of the salt content of the fixing medium might be taken as the basis of procedures designed to reduce these undesirable changes. It had previously been shown by Bauer and Ames² that the tissue reacts very differently to different solutions, depending on the particular salt present. This indicated that selection would be advisable in the salt used for the solution. However, we thought that control of the concentration of the solution would do much toward stabilizing the brain during fixation.

1. Hoch, August: Nerve Cell Changes in Somatic Diseases, *Am. J. Insan.* **55**:231, 1898.

2. Bauer and Ames: Contributions from the Third Division of the Neurological Institute, New York: June, 1914.

In attacking the problem, the plan followed was first to experiment with small bits of tissue weighing from 0.2 to 1.0 gm. These could be obtained easily, handled easily, and their reactions to the reagents determined quickly. A brief survey with various strengths of formaldehyd was first made. It was

TABLE 1.—*Fixation Experiments with 10 per cent. Liquor Formaldehydi and Varying Concentrations of Sodium or Copper Sulphate**

No	Disease	Salt	Percentage Concentration	Changes Made
1	General paralysis.....	Sodium sulphate	0.9 0.5 0.0	Two changes Twelfth day Seventeenth day
2	Arteriosclerosis.....	Sodium sulphate	0.9 0.5 0.0	Two changes Seventh day Tenth day
3	General paralysis.....	Sodium sulphate	0.9 0.5 0.0	Two changes Eighth day Twelfth day
4	Large cerebellar softening...	Sodium sulphate	0.9 0.5 0.0	One change Seventh day Fifteenth day
5	General paralysis.....	Sodium sulphate	0.9 0.5 0.0	32.54 and 30 c.c. of liquor formaldehydi added on second, fourth and fifth days respectively for percentage reduction determined by analysis Seventh day Tenth day
11	Arteriosclerosis.....	Copper sulphate	0.7 0.6 0.5 0.3 0.0	Third day Fifth day Seventh day Eleventh day
12	Dementia praecox.....	Copper sulphate	0.7 0.6 0.5 0.3 0.0	Second day Fourth day Eighth day Tenth day
13	General paralysis.....	Copper sulphate	0.7 0.5 0.3 0.0	Fourth day Sixth day Eighth day
14	General paralysis.....	Copper sulphate	0.8 0.5 0.0	24 and 30 c.c. of liquor formaldehydi added on second and third days respectively for percentage reduction determined by analysis Fourth day Seventh day
21	Dementia praecox.....	Control	...	Changed twice

* A standard of 3,000 c.c. was used as a fixing solution. The weights were taken after the brain had stood for twenty minutes, base down on a moist towel.

concluded that 10 per cent. liquor formaldehydi, U. S. P., as usually used was most satisfactory. Greater concentrations penetrated less rapidly, lesser concentrations caused too much mass increase. Alcohol combined with 10 per cent. liquor formaldehydi was tried, but no mixture of these was found which reduced the mass changes. In fact the rebound mass shrinkage was increased.

A number of salts in various concentrations were tried in combination with 10 per cent. liquor formaldehydi. These included magnesium sulphate, sodium

chlorid, sodium sulphate, ammonium chlorid and copper sulphate. Of these salts sodium sulphate and copper sulphate were most satisfactory, as they restrained swelling in lower concentrations than the others. The molecular content of the solutions of different salts seemed to bear no direct relation to their effects. With the same salt the effect, in restraining swelling, varied directly with the concentration. It was accordingly determined to limit further studies to these two salts.

It was next necessary to determine in what concentrations copper sulphate and sodium sulphate were most satisfactory. For this purpose a series of graded concentrations was made in 10 per cent. liquor formaldehydi; small bits of tissue

TABLE 2.—*Changes in Weight of Brains Described in Table 1.*

Brain	Neeropsy Weight	Maximum Weight	Minimum Weight	Fourth Week Weight	Weight of Portions* Removed for Examination (Estimated)
1	1,062	1,062	1,035 (14th day)	1,052	9 gm.
2	1,119	1,040 (7th day)	1,119 (35th day)	1,124	9 gm.
3	1,202	1,210 (5th day)	1,195 (20th day)	1,196	6 gm.
4	1,310	1,310 (1st day)	1,229 (12th day)	1,246	6 gm.
5	1,227	1,230 (11th day)	1,216 (4th day)	1,229†	6 gm.
11	1,228	1,245 (3d day)	1,192 (22d day)	1,195	6 gm.
12	1,193	1,222 (3d day)	1,172 (35th day)	1,174	5 gm.
13	1,220	1,259 (6th day)	1,220 (1st day)	1,240	6 gm.
14	1,319	1,340 (4th day)	1,319 (1st day)	1,330†	6 gm.
21	1,322	1,565 (5th day)	1,322 (1st day)	1,535	6 gm.

* The pieces removed for examination were taken before the fourth week, consequently their weights must be added to the weight of the fourth week for proper comparison.

† Taken at the end of two weeks.

TABLE 3.—*Maximum Changes in Weight of Brains*

Fixative	Brain	Maximum Gain or Loss in Gm.
Graded sodium sulphate in 10 per cent. liquor formaldehydi.....	1	-27
	2	-21
	3	+ 8
	4	-81
	5	-11
Graded copper sulphate in 10 per cent. liquor formaldehydi.....	11	-36
	12	+29
	13	+39
	14	+21
Ten per cent. liquor formaldehydi.....	21	+243

were fixed in these solutions and weighed daily during fixation, and for several weeks afterward. It was found that from 0.8 per cent. to 0.9 per cent. sodium sulphate* and 0.7 per cent. copper sulphate held the tissue most constant during its fixation period. On following this tissue over a longer period, however, it was found to steadily decrease in weight if left in the original fixative of 0.7 per cent. copper sulphate or 0.9 per cent. sodium sulphate in 10 per cent. liquor formaldehydi. Some pieces decreased as much as 35 per cent. of their original weight.

Having determined that for small pieces 0.9 per cent. sodium sulphate and 0.7 per cent. copper sulphate in 10 per cent. liquor formaldehydi was most

3. All percentage calculations were made on the basis of the anhydrous salt.

promising, it remained to try out these solutions in the fixation of whole brains, and to study brains, so fixed, microscopically for artefacts. It was foreseen that a single solution would not suffice to keep the brain weight constant, as the tissue behaves differently at different stages of fixation. It was thought that by reducing the salt concentration in steps, the brain might eventually be put in 10 per cent. liquor formaldehydi as its permanent preservative. Just when these changes would be necessary could be told only by trial with whole brains as the use of small pieces proved entirely unsatisfactory for this purpose. Consequently a series of brains was fixed in varying concentrations as shown in Table 1. In Table 2 are given the important data concerning changes in the weights of these brains.

DISCUSSION OF RESULTS

Examination of the tables of results shows that there is the same tendency toward swelling that appears in simple formaldehyd fixation, but in diminished degree. By the end of the first week with sodium sulphate solutions, and a little earlier with copper sulphate solutions, this swelling ceases and shrinkage begins. Weakening of the salt concentration at these times prevents this shrinkage to a large extent. While more gradual steps were made with some brains, it seems sufficient, with either salt to reduce first to a 0.5 per cent. solution and then to liquor formaldehydi 10 per cent. without salt. On the whole the sodium sulphate solutions stabilize the brains better than copper sulphate, but the copper solutions allow more rapid fixation.

Aside from the results of fixation, brains vary much as regards swelling and shrinkage, probably as the result of such factors as age, type of disease and the time that elapsed between death and the beginning of fixation. By the ordinary fixation method, the increase in weight of five brains observed during the period of swelling varied between 57 and 212 gm. In view of this individual variation, the following procedures are suggested as optimum stabilizing methods for all brains, rather than as perfect methods for any one brain:

1. Fix in 3,000 c.c. of sodium sulphate, 0.9 per cent.; liquor formaldehydi, U. S. P., 10 per cent. for 7 days. Then 3,000 c.c. of sodium sulphate, 0.5 per cent.; liquor formaldehydi 10 per cent.; for 3 to 5 days. Preserve in 3,000 c.c. of liquor formaldehydi 10 per cent.

2. Fix in 3,000 c.c. of copper sulphate, 0.8 per cent.; liquor formaldehydi, 10 per cent.; for 4 to 5 days. Then 3,000 c.c. of copper sulphate 0.5 per cent.; liquor formaldehydi 10 per cent.; for 2 to 3 days. Preserve, after short washing, in liquor formaldehydi 10 per cent.

In Method 1, two changes to fresh solutions should be made during the stage of 0.9 per cent. sodium sulphate. As an alternative, 30 c.c. liquor formaldehydi may be added on each of the second, third, fourth and fifth days.

In Method 2, 30 c.c. of liquor formaldehydi should be added on each of the second and third days.

From the standpoint of the gross appearance of the brain, the use of Method 1 leaves the brain with the same color as after simple formaldehyd fixation. Method 2, however, changes the color somewhat although the color washes out to some extent in the last solution. This method also makes the tissue firmer than does the first method.

Four stains, toluidin blue, hematoxylin, eosin and silver stains were tried on tissues fixed by these methods and were found to work satisfactorily.

The microscopic examination of the tissue for artefacts revealed an increase beyond the usual size of the perivascular and pericellular spaces. This feature was most prominent in the second layer, but occurred also deeper. While this is, of course, serious, it is probably dependent in part on the nature of the cases, i. e. the organic disease; for one case of dementia praecox was practically free from it. While the ordinary type of shrinkage alteration, the so-called sclerotic cell, was very infrequent, in two brains the closely related condition of crumbly or fragmented cytoplasm was present. In general, the nuclear detail was very good and the Nissl bodies were often demonstrable in the cells of the cortex of the frontal lobe.

In order to protect the surface of the cortex from the direct action of the formaldehyd, an area on Brain 4, was covered with glue. The glue became fixed and adherent to the surface. Pieces were then cut including this area and the adjacent uncovered cortex, and microscopic examination of these was made. Beneath the protected surface the cortex was a little paler than in the adjacent uncovered part. It resembled the sulcal cortex in this respect. A few dark shrunken cells were found in both areas.

CONCLUSIONS

1. It is desirable to stabilize brain tissue during the period of fixation.
2. Solutions of various salts affect brain tissue differently, independent of their concentration.
3. Sodium sulphate and copper sulphate have a strong restraining action on the swelling of brains during formaldehyd fixation.
4. Individual brains vary in their response to the same solution, hence a perfect fixing solution cannot be obtained.
5. One solution is inadequate for the whole period of fixation. Reduction of salt content as fixation proceeds is better.
6. The described methods are an improvement over simple liquor formaldehydi, 10 per cent., in that they tend to stabilize the tissue during fixation.

IMPLANTATION OF BACILLUS ACIDOPHILUS IN PERSONS WITH PSYCHOSES *

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HISTORICAL

In 1912, Berthelot and Bertrand ¹ described an organism which they isolated from human stools and called *Bacillus aminophilus*. Among other characteristics, it showed a marked ability to decarboxylate and deaminate amino-acids. Also, it was capable of producing histamine from histidine. Holmes and Rettinger ² alleged that dementia praecox was caused by the production of histamine by this organism in the intestinal tract.

From a study of the frequency of *B. aminophilus* in the stools of patients with dementia praecox, Jones ³ was unable to support the assumption of Holmes and Rettinger. He found: that this organism was present only in the proportion of from 1 to 20,000 to 1 to 100,000 of living bacteria; that it was found with a frequency usual in normal persons; that the histamine present was produced by other intestinal bacteria; that *B. aminophilus* did not utilize the carboxyl radical (COOH) of histidine if other sources of carbon were available; and that histamine (quoting Bayliss and Starling) is present in the normal intestinal mucosa. In short, Jones gives the impression that *B. aminophilus* bears no relationship to dementia praecox.

More recently, Cotton ⁴ has associated psychoses with focal infections, which begin at the teeth, and then spread to the stomach, duodenum and lower intestinal tract. Because of the frequency in such cases of the "Connellan-King diplococcus" and the colon bacillus, he suggests a causal relationship to the psychoses. In cases of lower

* From the bacteriologic laboratory and neuropsychiatric department of the Philadelphia General Hospital.

* Presented before the Philadelphia Pathological Society Nov. 23, 1922.

* Since this paper was submitted for publication, Kopeloff and Cheney have reported similar findings regarding the therapeutic effect of acidophilus milk, J. A. M. A. **69**:609 (Aug. 19) 1922.

1. Berthelot and Bertrand: Compt. rend. Acad. d. Sci. **154**:1826, 1912.

2. Holmes, Bayard, and Rettinger, J.: Lancet-Clinic **116**:145, 1916; Chicago Med. Rec. **38**:60, 1916.

3. Jones, H. M.: J. Infect. Dis. **22**:125, 1918.

4. Cotton, H. A.: New York M. J. **109**:397 and 454, 1919; The Defective, Delinquent and Insane, Princeton, N. J.: Princeton University Press, 1921.

intestinal infection he goes so far as to advocate colectomy, when this is necessary to eradicate the focus. He publishes reports of successful results following this procedure.

Metchnikoff introduced the ingestion of cultures of *Bacillus bulgaricus* in milk to eliminate intestinal putrefaction, on the assumption that the organisms and the acid of the milk cultures would be inimical to the putrefactive bacteria. Since then, however, it has been found that *B. bulgaricus* does not grow well in the intestine. This has led to other investigations of the possibility for elimination of intestinal putrefaction by changing the intestinal flora.

Rettger and Horton⁵ and later Hull and Rettger⁶ were able to change the intestinal flora of white rats from the ordinary type to an Acidophilic type by feeding milk or lactose. Torrey⁷ found this to be possible also in typhoid fever in man. Finally, Rettger and Cheplin,⁸ showed that it is possible to transform the intestinal flora of man in the majority of cases by the administration of broth cultures of *B. acidophilus*. In more refractory cases, the cultures were supplemented by the addition of lactose to the diet. Later, they introduced milk cultures of *B. acidophilus* and showed that then, the *B. acidophilus* content rose to 85 to 95 per cent of the cultivatable bacteria, and that this bacillus was able to proliferate in the intestinal tract.

In planning our work, we had in mind these points: if dementia praecox is caused by intestinal stasis and subsequent putrefaction, the implantation of *B. acidophilus* might tend to remove the putrefaction, and possibly, therefore, to alleviate the psychosis.

EXPERIMENTAL

In our series of experiments, ten patients were studied; four were carried throughout the study; the others were dropped from time to time as indicated in case histories. A normal person was used as a control for the rapidity, degree and permanency of *B. acidophilus* implantation.

Analyses of stools were made for one week prior to feeding with acidophilus milk to show: (1) the character of the stools; and (2) the presence or absence of acidophilic organisms. With the inception of the feeding, examinations of the stools were made daily until implantation was established: then the schedule was changed to two examinations per week to determine whether the implantations were being sustained.

5. Rettger, L. F., and Horton: Centralbl. f. Bakteriologie. **73**:362, 1914.

6. Hull, T. G., and Rettger, L. F.: J. Bacteriology. **2**:47, 1917.

7. Torrey: J. Infect. Dis. **16**:72, 1915.

8. Rettger, L. F., and Cheplin, H. A.: Treatise on the Transformation of the Intestinal Flora, with Special Reference to the Implantation of *Bacillus acidophilus*, New Haven, Conn.: Yale University Press, 1921.

The analyses consisted of: (1) staining smears by the gram method; (2) culturing fecal suspensions (in physiologic saline solution) on plates for numerical estimation of the *B. acidophilus* colonies; (3) culturing in litmus milk; and (4) culturing anaerobes in dextrose broth with the caustic soda-pyrogallic acid method.

The milk was prepared in bulk by heating in the autoclave at 5 pounds pressure for one hour. After cooling to room temperature, inoculations were made of about 2 c.c. of a twenty-four hour culture of *B. acidophilus*⁹ to each liter of the sterilized milk. This was incubated at 37 C. for fifteen to eighteen hours when it was sent to the

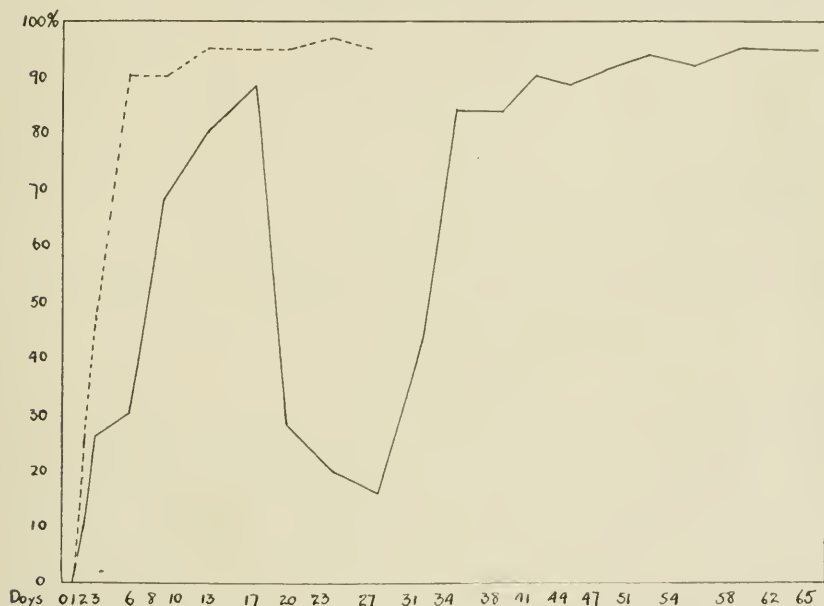


Chart 1.—Composite curve showing the percentage of *Bacillus acidophilus* in the stools during the study. Continuous line, patients with psychoses; broken line, a normal control.

wards for administration. The milk was fed for a period of sixty-four days. One liter a day was distributed for each patient, and was administered in three portions—one after each meal. No change in diet was made as the experiments of Rettger and Cheplin indicate that diet does not affect implantation. This treatment was never supplemented with lactose feeding, since implantation was secured without it. The methods employed were essentially the same as those advised by Rettger and Cheplin, except where it was necessary to make minor

9. Cultures of *B. acidophilus* were obtained through the courtesy of Dr. Leo. F. Rettger.

adaptations to the cases studied. Examination of the stools was continued for two weeks after the cessation of acidophilus administration, to determine how long the implantation persisted.

RESULTS

The analyses of the feces during the week prior to the administration of the acidophilus milk showed an absence of acidophilic bacteria in all patients. The stools were in general hard and well formed, and frequently showed the Welch bacillus. With the feeding of the milk, the stools remained negative for *B. acidophilus* until the second day, when this organism was recovered. The results have been plotted and are shown in composite form in Chart 1.

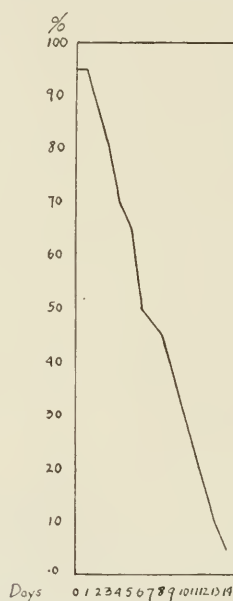


Chart 2.—Reduction in the numbers of *Bacillus acidophilus* after cessation of the feeding experiment.

It will be seen that the acidophilus organism made its appearance on the second day, and gradually increased in numbers until the seven-teenth day when an average of 88 per cent. was reached. At that time, the curve took a sudden drop and decreased for about ten days to a minimum of 17 per cent. The cause of this change of intestinal flora is problematic. We attributed the diminution to one of two reasons: either the milk was no longer fed, or the stools were kept too long before being delivered to the laboratory. We are inclined to believe that the milk was omitted from the patient's diet. Difficulty was encountered in obtaining the cooperation of our patients, and it is

possible that they disposed of the milk in some way other than by ingestion. The decrease in numbers of *B. acidophilus* continued for eleven days, when the curve began to rise and in a few days reached a maximum of from 90 to 95 per cent. where it remained for the duration of the investigation.

Bacillus welchii was no longer demonstrable in the specimens which contained *B. acidophilus* and the stools lost their putrefactive appearance. Milk cultures made from suspensions of feces were indistinguishable from twenty-four hour milk cultures of pure *B. acidophilus*, which indicates the completeness of acidophilus implantation.

We were unable to correlate any change in the mental condition of the patients with the feeding of acidophilus milk during the period of the experiment.

Chart 2 shows the persistence of *B. acidophilus* in our series. It appears that after two weeks' time, the recoverable number of *B. acidophilus* reaches a minimum of from 5 to 10 per cent. of the cultivatable bacteria; the administration of acidophilus milk must therefore be continued to maintain the implantation. The results obtained in Cases 4, 8 and 9 are of significance in another way. These three subjects had, at the beginning of our study, an acneiform eruption which improved with the implantation of *B. acidophilus*.

REPORTS OF CASES

The ten cases chosen for study all presented marked constipation and frequently complained of headache and fatigue. One case showed marked secondary anemia. Several had a wide spread acneiform skin eruption. Urinalyses were negative with the exception of positive indican reactions in Cases 1 and 9. Nothing of importance was noted in the blood counts except the usual tendency toward a mild lymphocytosis. The basal metabolism rate in Case 5 was reported as + 24. Spinal fluid findings were negative. Blood urea determinations were normal. All Wassermann reactions were negative.

No changes were made in the routine ward diet during the feeding of acidophilus milk. The patients, with a few exceptions, did not complain of this procedure although toward the end of the period of feeding, several expressed some discontent at the large quantity of milk that had been offered them. Other ward patients with similar mental states were used as controls.

CASE 1.—E. S., a girl, aged 15, was admitted Sept. 29, 1921, and was discharged in improved condition May 25, 1922; the diagnosis made was: dementia praecox, catatonic type.

The patient was excited, combative, and refused to eat, asserting that her food was poisoned. Her conversation at times was incoherent and she exhibited verberation. There were stereotyped movements and periods of causeless laughter; auditory hallucinations also were present.

Course.—Periods of excitement alternated with periods of stupor, in which catalepsy, with its characteristic spring resistance and rigidity, were observed. Gradual improvement began after three months' stay in the hospital.

The patient was given acidophilus milk throughout the entire period of the experiment from February 5 to April 8 inclusive. This was associated with alleviation of constipation, and for three days there was a tendency to diarrhea. The patient gained twelve pounds during this period of feeding. We cannot be sure, however, that the mental improvement was associated with this procedure; she was considered a favorable case from the time of admission. Two months after discharge, when examined in the outpatient department, there had been no return of constipation, and the patient showed continued improvement. She had only partial insight into her mental condition.

CASE 2.—V. S., a married woman, aged 32, was admitted Jan. 9, 1922, and was discharged improved April 30, 1922. The diagnosis was: schizophrenic reaction.

When admitted, the patient smiled and laughed in typical fashion; she was very obscene and untidy; experienced hallucinations, and at times was resistive. She showed a definite ambivalent tendency towards her husband (both love and hate) and had poor insight.

Course.—There was little improvement during the first two months in the hospital; later, the hallucinations disappeared, and periods of excitement terminated quickly after the use of continuous tubs. She began to show more interest in her surroundings, and cooperated in occupational therapy. At the time of discharge, she had only partial insight into her condition.

This patient was given acidophilus milk throughout the experimental period from February 5 to April 8 inclusive. One day she showed a tendency toward diarrhea. She lost five pounds during the period of observation. However, she had a tendency to obesity, and was in much better condition physically at the time of discharge; the constipation was definitely relieved.

CASE 3.—A. B., a married woman, age 27, was admitted Jan. 14, 1922, and the diagnosis was: schizophrenic reaction. She improved and was discharged April 4, 1922.

The onset followed desertion by her husband when she became indifferent, apathetic, and at times impulsive. She complained of feeling doped and that people read her mind, though there were no hallucinations. She was in fair physical condition.

Course.—The course was characterized by gradual improvement under treatment up to the time of discharge, when the patient had good insight into her condition. Acidophilus feeding in this case likewise resulted in the elimination of constipation, and an improvement of the general physical condition. The patient gained five pounds during the period of feeding, from February 5 to 16 when she was transferred from the department.

CASE 4.—M. C., a single woman, aged 25, was admitted Jan. 17, 1922, and discharged improved Feb. 28, 1922. The diagnosis was: schizophrenic reaction. The onset of the psychosis was in June, 1921, following the loss of her position. The patient developed ideas of reference, became silly and was antagonistic toward a sister. She frequently refused to eat, because the "food was poisoned." At times, she was untidy and childlike. She experienced hallucinations and had ideas of death and vague sexual phantasies. She presented a typical seclusive personality make-up.

Course.—Gradual improvement occurred during the last three months in the hospital. She was taught facts concerning sexual matters, and became less jealous of her sister; she had excellent insight at the time of her discharge when arrangements were made for return to her native country.

The patient was fed acidophilus milk throughout her stay in the hospital with resultant elimination of constipation. An acne-like eruption disappeared. The treatment in this case was considered to be very helpful, and an excellent adjunct in the treatment which was instituted from the psychogenetic viewpoint. The acidophilus milk was administered from February 5 to April 8, inclusive.

CASE 5.—A. W., a married woman, aged 47, was admitted Jan. 15, 1922, and discharged May 20, 1922, improved. The diagnosis made was: manic-depressive insanity (atypical manic phase).

On admission the patient showed marked pressure of speech and activity interspersed with periods of anxiety and depression. She had been running around the street in her night clothes; was very noisy and at times, destructive. She was frequently observed in the act of burning old clothes in her yard, and neighbors were apprehensive lest she set fire to her property.

Physical Examination.—Revealed a thyroid toxicosis; lobectomy had been performed before admission. The Goetsch epinephrin test was negative; the pulse averaged from 90 to 100; the basal metabolism rate was +24; all eye signs were positive; typical fine tremors of the hands were present; but there was no loss of weight. The patient was sent to a surgical ward, but the consulting surgeon stated that operation was not indicated.

Course.—During her stay in the hospital she showed little improvement. She presented a picture of an atypical affective disorder, with predominance of a manic reaction. Acidophilus feeding resulted in a gain of three pounds, but no other change was noted. Constipation had never been a troublesome factor. Acidophilus milk was administered from February 5 to April 8, inclusive.

CASE 6.—A man, aged 23, laborer by occupation, was admitted Feb. 1, 1922. He was adjudged insane April 29, 1922, as he had not improved. The diagnosis was: dementia praecox of catatonic type.

This patient showed a typical catatonic reaction, characterized by negativism, complete mutism and catalepsy. He frequently required tube feeding. His physical condition was good with the exception of general undernutrition and marked constipation.

Course.—The course was characterized by no improvement. Feeding of acidophilus milk resulted in clearing up the skin condition and prevented further emaciation; in spite of irregular and enforced nourishment, he did not lose weight. Acidophilus milk was administered from February 10 to March 28, inclusive.

CASE 7.—F. W., a man aged 34, was admitted Jan. 8, 1922, and discharged April 27, 1922, without improvement. The diagnosis was: dementia praecox.

A history was given of two previous confinements with a similar mental disorder. The patient maintained that he was Jesus Christ; a creator, and had unusual powers to control the destiny of the universe. He spoke of occult phenomena and gave frequent dissertations on the cosmos and theosophical matters. He had some paranoid ideas, centered about a certain individual whom he blamed for his father's financial ruin.

Course.—No change was observed in the patient's mental condition during his stay in the hospital. He never experienced hallucinations and showed no

change in the delusions mentioned. He had no insight into his mental condition. Physically he was well except for marked constipation. He obtained relief from this latter symptom, and gained in weight, after the administration of acidophilus milk, which was given from February 5 to April 8, inclusive.

CASE 8.—D. T., a man, aged 25, was admitted Jan. 19, 1922, and was committed March 15, 1922, as he had not improved. The diagnosis was: dementia praecox.

The onset of the psychosis was in September, 1920, when the patient began talking and smiling to himself and became impulsive and quarrelsome with his associates. He later developed gonorrhea and while under treatment became very excited and experienced hallucinations. The excitement alternated with periods of mutism and catatonia; at these times he refused to eat, and required tube feeding.

Course.—The patient continued to be apathetic and resistive, with periods of stupor and required tube feeding. At the time of commitment, no mental improvement was noted. Acidophilus milk was given irregularly to this patient. However, a marked change appeared in the acneiform eruption on his face and back, which cleared up considerably. His weight remained stationary. Other patients in the ward in similar condition showed a tendency to lose weight. Acidophilus milk was administered from February 5 to March 21, inclusive.

CASE 9.—R. S., a woman aged 36, was admitted Jan. 10, 1920, and discharged Feb. 18, 1920. The diagnosis made was: delirious reaction.

On admission the patient was very apprehensive and was experiencing visual and auditory hallucinations. She was agitated, restless, resistive and disoriented. During the first month in the hospital, she developed paranoid ideas about the physician who performed the lumbar puncture. Physically, she showed: marked secondary anemia; positive indican reaction; a trace of albumen in the urine, and marked constipation. A marked acneiform eruption was present over the face and back. Acidophilus milk feeding resulted in the acneiform eruption clearing up entirely. She gained ten pounds in weight and constipation was alleviated. The patient had diarrhea on one day during the period of treatment. At the time of discharge, she had from one to two normal bowel movements daily. Acidophilus milk was administered from February 5 to February 21, inclusive.

CASE 10.—G. B., a man aged 37, foreman by occupation, was admitted Jan. 30, 1922, and was committed March 13, 1922, as he had not improved. The diagnosis made was: depressive reaction. The patient was tearful, agitated and expressed many hypochondriacal complaints, with marked self-condemnation. He had attempted suicide by hanging before admission. The onset of the trouble was a reaction to financial reverses, domestic infelicity, and change of position. Acidophilus milk was administered from February 21 to March 10, inclusive.

COMMENTS

The chief aims of our experiments were: first, the relief of constipation by implantation and maintenance of *Bacillus acidophilus* in the intestine; and second, observance of the effects on the course of the psychosis. Constipation, with or without putrefaction, is frequent in patients with psychoses. When the sedentary life and frequent food refusal of these patients is considered, constipation may obviously be a result or a concomitant of the psychosis. The constipation, with what-

ever putrefaction there may be, can be corrected by *B. acidophilus* implantation and this condition may be maintained. In our series of cases this change of intestinal flora was not associated with a change in the mental condition.

We think that Holmes and Rettinger have not demonstrated an etiologic relationship between *B. aminophilus* fermentation and dementia praecox. We made no tests for the presence of *B. aminophilus*, but after the establishment of *B. acidophilus*, the few colonies of other organisms that appeared in cultures were not of the aminophilus type. We do not believe that colectomy, advocated by Cotton, is justifiable. Putrefaction, if present, may be eliminated by a change in the intestinal flora. Although the administration of acidophilus milk did not improve the mental condition of our patients, we believe that it may be employed with benefit during a psychosis because of the resultant relief of constipation, the improved appetite, and the gain in weight.

CONCLUSIONS

1. Implantation of *Bacillus acidophilus* in the intestine of patients with psychoses by the oral administration of acidophilus milk resulted in elimination of intestinal putrefaction and in some cases in an increase in weight but no improvement in the mental condition.
2. Intestinal putrefaction is probably a concomitant of some forms of psychosis if it is at all related.
3. The administration of acidophilus milk is recommended in the treatment of psychoses as a means to physical betterment.

Obituary

EDWARD MERCUR WILLIAMS, M.D.

1881-1923

In the death of Dr. Edward Mercur Williams, the American Neurological Association has lost one of the most promising of its younger members, one who bade fair to become a leader in American neurology. It is fitting that I should place on record an estimate of his acquirements and character as, for three years, from 1909 to 1912, he was my private assistant and associate, and was also a valued member of the neurologic staff of the medical school of the University of Pennsylvania. Thoroughly trained in general medicine and in neurology, honest in his convictions and methods, he was courteous, kindly and unobtrusive. He was a gentle soul, although one who, when necessary, could stand firmly for his own position and opinions. He became a member of this association in 1912 and contributed several valuable papers to our proceedings.

The parents of Dr. Williams were Horace Guernsey Williams and Ann Bowkley Williams. He was born October 16, 1881, at Wilkes Barre, Pa. From Wilkes Barre, the family moved to Lykens, Pa., situated in a region noted for a highly prized variety of coal. For several years, his father, a distinguished mining engineer, was interested in operations in the Lykens Valley for the Pennsylvania Railroad. While at Lykens, Dr. Williams went to the neighborhood school.

In 1895, he went with his family to St. Davids, Pennsylvania, and became a student at the Radnor High School, where he graduated in 1900. After taking a special course in biology in the University of Pennsylvania, he entered the medical school, graduating with a good record in 1905. Soon after graduation he became an intern in the Monongahela Surgical Hospital, in the western part of Pennsylvania, where he remained until some time in 1906. After leaving Monongahela, he came to Philadelphia and took a postgraduate course in the Polyclinic Hospital School.

In 1907, he went abroad to complete his medical studies and selected Vienna as the first stopping place in his foreign postgraduate pilgrimage. Here he remained for almost two years, working in the laboratories of Professor Obersteiner, Frankel Hochwart, Marburg and Ludwig Braun. While in Vienna, he became highly expert in the cutting, staining and studying of specimens illustrating diseases of the



EDWARD MERCUR WILLIAMS, M.D.

nervous system. He completed his foreign studies in Paris, chiefly under the direction of Pierre Marie. In Paris, he also made general use of the clinics, hospitals and societies.

While abroad, Dr. Williams published several papers, two in German,—“Vergleichend anatomische Studien über den Bau und die Bedeutung der Oliva inferior der Säugetiere und Vögel” (Concerning the Anatomic Structure of the Inferior Olive in Mammals and Pigeons); and “Ueber Fazialislähmung nach Zahnextraktion” (Facial Paralysis Following Extraction of the Teeth).

The first of these papers was written under the stimulus and supervision of Professor Obersteiner, and the second was from the Vienna Medical Clinic of Professor v. Noorden. Both of these contributions contained original observations of sufficient value to merit the approval of the distinguished group of neuropathologists of Vienna in whose laboratories Dr. Williams spent most of his working hours.

Dr. Williams returned to this country in August, 1909, when I first made his personal acquaintance. He soon became a member of the neurologic staff of the medical school of the University of Pennsylvania, in which he held several important positions, among these being, Instructor in Neuropathology, Assistant Neurologist and Assistant Physician in the neurologic dispensary to the hospital of the University of Pennsylvania. In all of these positions he did excellent work. During his connection with the University Medical School, he published several important papers—“Chronic Hypertrophic Spinal Pachymeningitis” (with Dr. Charles K. Mills), “État Vermoulu,” “Intermittent Claudication in the Upper Extremities,” “Cerebral Rheumatism” and “Typhoidal Hemiplegia.”

Dr. Williams moved to Vinita, Okla., in November, 1912, becoming the resident physician of the East Oklahoma Hospital for the Insane. Here he did excellent work for about one year, after which he removed to Sioux City, Iowa. In Sioux City, he engaged in private work, chiefly as a neurologic consultant, devoting himself assiduously to practice, research and clinical investigation. In addition to his work as a neurologic consultant, he became well known in the courts as a medical witness in cases involving insanity and traumatic and other affections of the brain and nervous system. Dr. Williams soon became well known throughout the middle west, his medical work extending to various localities in Iowa, Nebraska, Minnesota and South Dakota.

In the preparation of this sketch, many letters of condolence and of appreciation from members of the medical and legal professions have come under my notice. These are of the same general tenor. The letters from his medical confreres lay stress on his thoroughness, efficiency, honesty and reliability. They speak of the constant use

made by him of the technical knowledge obtained by him in the laboratory. The tributes from the legal profession all call attention to his modesty, sincerity and learning, and to the clearness, precision and truthfulness of his testimony. His standing in the courts as testified to both by members of the bench and the bar was of the highest character.

Dr. Williams' career in Sioux City extended over a little more than nine years.

His health was never very good. He had some form of cardiac disease, and while he was fond of outdoor life, he was compelled to restrict his exercise.

In January, 1923, he had an attack of influenza, which was followed by pneumonia. He went to St. Joseph's Mercy Hospital in Sioux City, and although he received every attention from his medical brethren and others connected with the hospital, he died January 5, 1923. His death not only left a void in his family and among his friends, but was the subject of marked regret among the profession of Sioux City and the state of Iowa. Resolutions of appreciation and regret were adopted by the Iowa State Medical Society.

During the short period of about fourteen years between the time of his return from Europe and his death, besides the contributions already mentioned Dr. Williams published a considerable list of papers, some of which were of unusual value, both from a clinical and pathologic standpoint.

In 1911, he married Florence Henry Lofland, of St. Davids, Pa. He is survived by his devoted wife and one child, Sarah Lofland Williams, born in 1916.

When one in the fulness of years departs from our midst, we may have regrets that we can no longer be cheered by his presence, and in that presence can no longer be stimulated by the story of his well wrought career; but when the passer is one stricken when he had scarcely reached his prime, one who gave promise of much to come, then are our hearts filled with a peculiar sadness, begotten by the thought that he was cut off too soon for him to achieve his soul's desires, and yet, we are gladdened when we feel that such opportunities as he had were not neglected and that the results he obtained are worthy of our admiration.

CHAS. K. MILLS.

News and Comment

THE THERAPY OF EXOPHTHALMIC GOITER

Exophthalmic goiter has long offered a problem of serious import to the clinician, since it causes much immediate distress to the patient and presents uncertainties of prognosis in most cases. Until recently, errors in diagnosis were frequent, for few clearly defined aids to clinical judgment were available. Now that there is more agreement in respect to the distinguishing characteristics of exophthalmic goiter, the therapy of the disease has become the subject of active discussion. In a recent issue of the *Archives of Internal Medicine*¹ is presented the outcome of serious researches by competent investigators which emphasize the conflicting points of view.

The crux of the situation involves the natural or spontaneous course of the disease. This is admittedly fundamental to any final evaluation of specific measures of treatment that may be undertaken. It is often alleged that the natural course of exophthalmic goiter is a most uneven one, and that improvements and relapses may occur at any time spontaneously. Means and Holmes of Boston have wisely remarked, however, that until some one produces a satisfactory experimental exophthalmic goiter, controls will be secured only by chance. Under such circumstances, therefore, it will not be easy to withhold promising treatment from patients, even if a considerable element of uncertainty as to its fundamental value may exist.

Recent treatment of exophthalmic goiter has for the most part proceeded on the theory that it really represents an aspect of hyperthyroidism. Means and Holmes remark that if we accept the theory that the symptoms manifested in hyperthyroidism are due to increased activity of the cells composing the thyroid gland, then the application of any method of treatment which is known to inhibit or destroy cell function would be rational. Surgery and roentgen-ray treatment have accordingly vied with each other for favor in bringing about the desired result. Rarely has it been possible to study such an array of carefully collected data as the intelligent collaboration of the internist, the surgeon and the roentgenologist has made possible in the study of a large number of patients at the Massachusetts General Hospital by Means and his collaborators. About two thirds of these cases exhibiting unmistakable exophthalmic goiter showed either recovery or improvement coincident with treatment by suitable irradiation with roentgen rays. The Boston clinicians assert that in exophthalmic goiter, when treated by the roentgen ray, if good results are not secured in a few months, surgery should be employed. Prolonged roentgen-ray treatment in patients showing no response is undesirable. They also believe that some patients with exophthalmic goiter who are not

1. Means, J. H., and Holmes, G. W.: Further Observations on the Roentgen-ray Treatment of Toxic Goiter, *Arch. Int. Med.* **31**:303 (March) 1923. Kessel, Leo; Lieb, C. C., and Hyman, H. T.: Studies of Exophthalmic Goiter and the Involuntary Nervous System: Kessel, Leo; Hyman, H. T., and Lande, Herman: III, A Study of Fifty Consecutive Cases of Exophthalmic Goiter, *ibid.* **31**:433 (March) 1923.

cured by the roentgen ray are, perhaps, made better operative risks by it. A combination of the two forms of treatment may sometimes accomplish more than either does alone.

In contrast with this is the report of the course of fifty cases of fully developed exophthalmic goiter observed at the Mount Sinai Hospital, New York, by Kessel, Hyman and Lande. The course of the illness was followed with as little interference with the natural tendency of the disease as was compatible with the comfort of the patient. At any rate, "specific" therapeutic procedures were not instituted. Such observations on patients kept essentially at rest alone may quite properly be regarded as a control investigation for comparison with the result attributed to irradiation and surgery. In the opinion of the New York clinicians, the spontaneous course of exophthalmic goiter is toward arrest in the vast majority of cases. In patients who develop the disease late in life (after 45 or 50) the prognosis is poor. If these cases are excepted, the prognosis is excellent under a regimen of "skilful neglect." To establish the efficacy of any specific therapeutic measure, we are told further, one should demand that definite proof be offered that the results obtained are better than those reported here of the "spontaneous" course of the disease.

All investigators now agree that until a specific diagnostic test is discovered, no reported case of exophthalmic goiter should be accepted as a genuine case unless the basal metabolism is distinctly and repeatedly elevated. According to the New York clinicians, by this alone can the disease be differentiated from "autonomic imbalance." In the stage of arrest, differentiation can be made only by the history of the crisis. The clinical picture varies as the age of the patient at the time of onset of the disease. In other words, the variation, especially in regard to the presence and extent of the exophthalmos, is dependent, we are told, on the end tissues of the patient, rather than on the specific elaboration of any toxic product. It may be well to reiterate here that thyroid hyperplasia and thyroid adenoma may exist for years without at any time causing sympathomimetic symptoms or alteration in metabolism. Kessel, Hyman and Lande insist that in exophthalmic goiter the dominant derangement is in the realm of the involuntary nervous system. This may not be primary; but the primary cause, whatever it is, must at least operate through the mediation of the involuntary nervous system. Means and Holmes point out, further, that, in toxic adenoma, roentgen-ray treatment appears to effect a similar improvement to that noted by them in exophthalmic goiter; but so far they have used irradiation only with patients who have refused operation. In toxic adenoma, in contrast to exophthalmic goiter, surgery probably removes the actual cause of the disease, the adenoma. The indication for surgery, they add, would, therefore, seem more definite than in exophthalmic goiter. Even in toxic adenoma, however, in certain cases that are too thyrotoxic for safe operation, the roentgen ray may be used to advantage. In the light of present-day evidence, the choice of therapeutic procedure presents, indeed, a difficult perplexity.—*Jour. A. M. A.*, April 14, 1923.

Abstracts from Current Literature

RESEARCHES ON THE ABDUCENS NERVE AND PARTICULARLY
ON THE SIGNIFICANCE OF ITS ACCESSORY NUCLEUS. T. TERNI.
Folia Neuro-Biolog. **12**:277, 1922.

The author refers to the first researches of Lugaro and Pacetti after whom the "nucleus accessorius abducentis" was named, and to the work of later investigators: Siemerling and Boedeker, Giannuli, Panegrossi, Van Gehuchten, Kaplan and Finkelburg, Bach, Tsuchida, Von Falkenburg, Jacobsohn, Kappers, Cajal, Fuse, and Sterzi, whose conclusions on the existence of an accessory nucleus of the sixth nerve in vertebrates are contradictory.

In order to clear up the morphology and possibly the functional significance that the accessory nucleus possesses in the constitution of the sixth nerve, the author has undertaken the investigation reported in this paper. He has endeavored to find first, the central origin of the abducens in the representatives of those groups of animals (fishes, reptiles) in which no duplicity of the nucleus has been demonstrated; and secondly, to define with more detail than has been done by others, the constitution and the central relations of the sixth nerve in the representatives of animals (birds, mammals), in whom scanty observations had led to positive preliminary results. His material for investigations were mainly embryos in which the application of Cajal's method for neurofibrils gave excellent results.

For the researches on the trout, the swimming embryos of *Salmo fario* and *Salmo carpio* from 10 to 20 mm. long, were used; serial sections were cut transversely.

For the investigation in *Gongylus ocellatus*, there were utilized ninety-seven embryos in different stages of development from the very early periods up to birth, cut in series in different planes. The greater number of embryos corresponded approximately to the same stages already described by the author in one of his works on the spinal cord. Besides, a few series of embryos fixed in Zenker's fluid and stained with alum-carmin were made. Serial sections of brains of new-born *Gongylus* stained with Cajal's method, and of adult *Gongylus* stained either with Cajal's, or with Weigert's or Nissl's methods were also prepared.

For the investigations on chickens, several embryos at different stages of development, impregnated with Cajal's method, were used. One series of chicks was prepared with the Cajal method and one with the Weigert method. Adult animals were also dissected for the purpose of becoming oriented on the muscles supplied by the sixth nerve.

The observations on *Mus rattus* were conducted on embryos, whose brains were cut transversely and longitudinally and stained with Cajal's method.

The results of the author's investigations on the central origin and on the peripheral distribution of the sixth nerve are preceded by the most important bibliographic data on the subject. Also, some personal contributions to the development of the nerve, and some information concerning the morphology of the third eyelid (an organ which is directly or indirectly operated by muscles supplied by the abducens), are given.

The findings of the author are:

1. *Fishes*.—Trout: Cajal's and Tello's findings on the positions of the nucleus of origin of the abducens are confirmed. An accessory nucleus of the abducens, situated laterad and distant from the principal nucleus does not exist. A third eyelid and a muscle retractor of the ocular bulb, differentiated as a special muscle, is also lacking. The abducens in its peripheral distribution is clearly seen to reach a single muscle, the external rectus.

2. *Reptiles*.—Gongylus: In embryos at the stage 4 (corresponding to stage 123 of Peter in *Lacerta a.*) the principal nucleus of the abducens is situated, as in all tetrapoda, very dorsad in the bulb, almost in contact with the ventral and lateral surface of the fasciculus longitudinalis medialis. It forms a small continuous column, which goes from a level corresponding to the caudal pole of the motor nucleus of the fifth nerve, to a point very near to the rostral pole of the motor nucleus of the facial. The cells are fairly large, appear like stars, having dendrites more developed outwardly, and which stain poorly with silver. The neurites describe a slight arch thus causing the root of the abducens to emerge somewhat far from the middle line. Not all the radicular fibers originate from the cells of the principal nucleus. In the root of the sixth nerve one may clearly see fibers that come from another region of the rhombencephalon. These fibers are very large; they run in bundles or singly and form an elbow in correspondence with the principal nucleus, and then follow the same direction as the nerve in their common exit from the rhombencephalon. These fibers originate from a group of cells very characteristic in shape, which are situated in a ventrolateral position, mediad to the descending root of the trigeminal. This nucleus, never described in reptiles, corresponds to the one described by Van Gehuchten in chickens and by Lugaro in rabbits. It is a small flat column, which, from a caudal level corresponding to the rostral pole of the motor nucleus of the facial, goes a short distance below the caudal pole of the principal motor nucleus of the trigeminal. This nuclear column has approximately the same length as the principal nucleus; it is displaced slightly caudad. The cells, owing to their very typical form, are easily distinguished from the cells of the principal nucleus and from those of the other nearby nuclei. They are multipolar, very large, larger than those of the principal nucleus, and are very easily stained with Cajal's method. They present two principal groups of fine long dendrites and an axon which usually originates from the dorsal and median portion of the cell in the proximity of the nucleus, which is very large and provided with a conspicuous nucleolus.

The cells of the accessory nucleus form a very thick and well circumscribed group in the embryo. They cannot be mistaken for other near or distant elements, such as the cells of the sensory nucleus of the fifth, the cells of the nucleus of Deiter and the elements of the nucleus magnocellularis of Cajal—the latter possessing an entirely different aspect, size and distribution. The axis-cylinders run in bundles of two to four and, before entering the common root of the sixth nerve, describe an elbow around the principal nucleus. They cross the fibers of the direct vestibulospinal tract and enter, without association, among the fibers of the principal nucleus; they maintain their independence from other systems of fibers, such as the trigeminal system and the central acoustic pathway. A conspicuous group of dendrites of the cells of the accessory nucleus of the abducens, however, constantly converges toward the descending root of the trigeminal, and penetrates among its fibers. It is

likely that the accessory nucleus and the principal nucleus of the sixth nerve originate from a cell group which originally constituted a single unit.

In very advanced stages, up to birth, the transformations in the general constitution of the nucleus of the sixth nerve are very slight. At birth, the relationship between accessory and principal nucleus remains the same as in embryos, except for the neurofibrillar reaction which is less distinct. In the adult this relationship is not so easy to trace, since the Cajal method does not stain this system very electively. With the Weigert method the accessory radix of the sixth nerve is seen entering the common root, but its fibers cannot be followed to their origin from the accessory nucleus. With alumarcarmine and the Nissl method the small group of cells of the accessory nucleus is found in the same position as in the embryo, namely in proximity to the descending radix of the fifth and slightly caudad to the principal nucleus of the sixth. However, the cell elements cannot be well differentiated from other systems nearby.

Here the author gives the peripheral distribution of the abducens, studied in correlation with its central configuration. He found that in *Gongylus*, the muscles supplied by the abducens correspond almost entirely to those described in *Lacerta* by M. Weber. For this study he used the Cajal method in embryos of very advanced stages—from stage 4 on. After a clear and complete description of the muscles and their nerve supply, he gives the number of fibers found in the two nerve trunks which result from the bifurcation of the abducens as 150. One hundred of these are for the external rectus, and thirty to forty fibers compose the nerve for the motor and retractor muscles of the nictitating membrane. There seems to exist a correlation between the number of the fibers of the two branches of the abducens and the number of the cells of the two nuclei of origin. The author is of the opinion that the fibers originating from the accessory nucleus are distributed to the muscles of the third eyelid (retractor and bursal), and that the other fibers from the principal nucleus, are for the external rectus. A table giving the number of cells of the nuclei and the corresponding fibers in the main trunk, and in both branches as distributed to the different muscles, in the different stages, is given. The number of cells of the accessory nucleus do not change at the different embryonal stages.

3. *Birds.—Gallus domesticus.* In embryos of chicks of 6 or more days, the accessory nucleus of the abducens is made up from a thick group of cells smaller than the principal nucleus, situated in the medulla oblongata at about the same level as the principal nucleus. It extends from the caudal pole of the motor nucleus of the facial to about the rostral pole of the dorsal nucleus of the vagus and glossopharyngeal. It is located between the medial margin of the descending root of the trigeminal and a cellular group of still scarcely differentiated elements, which the author believes to be the superior olive. The cells of the accessory nucleus are multipolar, much elongated, and possess dendrites which converge in a conspicuous group toward the lateral surface of the bulb, where the fibers of the descending root of the trigeminus course. The cells are somewhat smaller than the cells of the nucleus magnocellularis reticularis. The neurites of many reticular cells, situated in the proximity of the accessory nucleus of the abducens, approach, or cross, the direction of the accessory root of the abducens. For this reason some authors have mistaken the nuclei of the reticular substance, viz: the superior, the middle, the inferior and the disseminated, for the accessory nucleus. In more advanced stages up to the thirteenth day, the accessory nucleus becomes more con-

spicuous, but its relationship does not vary. In a series of adult chickens prepared with the Weigert method, the author has identified the nucleus in a small column of cells at the level of the principal nucleus of the sixth, near the external margin of the superior olive, and almost in contact with the medial edge of the spinal root of the fifth. In general, the accessory nucleus of the abducens and the fibers which originate from it, show an appearance similar to the formation indicated with (a) in the Figure I of Cajal (1908). Regarding the distribution of the abducens, the author confirms what has been found in the adult. In embryos of 6 days, one can recognize a distinct muscular rudiment for the external rectus and for the muscles of the nictitating membrane, and the branches of the sixth pair which supply the separate rudiments.

4. *Mammals*.—*Mus Rattus*. Extensive investigations on the accessory nucleus of the sixth nerve could not be carried out, because in the embryos of these animals the Cajal reaction does not give as good results as in reptiles and birds. The accessory nucleus of the abducens is made up of a thick cell group, well separated from the motor nucleus of the facial and from the principal nucleus of the abducens, situated at about the same level as the latter but much more ventrad and laterad. Its position is between the nucleus of the facial and the principal nucleus of the abducens, but much nearer to the former, and well distant from the descending root of the fifth. The cells are stellate, long, but smaller than the cells of the substantia reticularis grisea.

The results of some simple physiologic experiments on the trigeminal-abducens reflex, obtained by the author, harmonize with the anatomic findings. In reptiles, a very brisk reflex of the third eyelid is found. It consists in the immediate closure of the lid on mechanical or electrical stimulation of the peripheral territory of the trigeminal. At the same time a slight enophthalmos is observed without rotatory movement of the ocular bulb. After decerebration and destruction of the optic lobes, the reflex persists unchanged. In chickens and in other domestic birds the reflex has also been found by the author, but it is not accompanied by exophthalmos. (These experiments were reported in *Archivio de Fisiologia*, 20:4, 1922.)

In discussing the morphologic and functional significance of the sixth nerve and its accessory nucleus, the author concludes that although this nerve, in a large number of vertebrates, supplies more than one single muscle, and, although it originates from a nuclear complex whose accessory constituent is situated quite laterad and ventrad in the medulla oblongata, yet it presents the singular appearance of a ventral spinal root. The accessory nucleus of the sixth nerve supplies those muscles which either directly or indirectly are connected with the movements of the third eyelid. The grouping of the radicular cells of the abducens in two nuclei, distinct and very far from each other, is in perfect accord with the doctrine of Kappers, according to whom a motor nucleus tends to approach the system from which it receives (or will receive later) the greatest amount of functional stimulus. It is likely that the accessory nucleus will make important connections with the different pathways making up the descending root of the trigeminal, inasmuch as the movement of the nictitating membrane is closely related to stimuli from the territory of the peripheral distribution of the trigeminus. The principal nucleus remains, for obvious reasons, near the systems of the optic and acoustic pathways, a portion of which run in the medial longitudinal fasciculus.

SPIROCHETES IN SYPHILITIC ENDARTERITIS OF THE BRAIN.

F. SIOLI, Arch. f. Psychiat. u. Nervenkrankh. 66:318 (Oct. 18) 1922.

In this article Sioli discusses the general problem of so-called metasyphilis, his general thesis being that it is premature to make any very definite theoretical assumptions until more work has been done to show the relation or non-relation of the spirochetes to the pathologic processes. He first calls attention to the fact that the idea of general paralysis as a syphilitic disease depending on the effect of syphilitic toxin without the spirochetes in the brain, was upset by the work of Noguchi and Moore and their followers. In cases of endarteritis of the small cortical vessels, spirochetes have not been found in the past, which led to the hypothesis that the endarteritis was the result, not of the local spirochetal activity, but of a toxic process. This has been the general idea of the metasyphilitic processes and therefore it becomes of great importance to determine the presence or absence of spirochetes in cases of endarteritis which will again illuminate the whole metasyphilitic problem.

The endarteritis of the small brain vessels was first described by Schüle in 1872 and its histologic characteristics were established in 1903 by Nissl on the basis of four cases, three of which had been observed by Alzheimer and discussed under the title "Diffuse Brain Syphilis Without Adventitial Sheath Infiltration" or again as "A Non-Gummatous Form of Brain Lues." Nissl characterizes the process as follows: The change is diffuse; adventitial sheath infiltration does not occur except for an occasional mast-cell. The cells of the intima are greatly proliferated and there is considerable increase of the cells of the intima which may divide the vessel lumen into several channels. As a result of the proliferation of the endothelial and adventitial cells, the muscle coats are disorganized. New vessels are formed by outgrowths of the old, and the elastic coat is broken up. There is a relatively small amount of disorder of the tangential fibers and supraradial network. Glia proliferation occurs, but of a type different from that found in general paralysis, namely, a growth of the protoplasm with very little fiber formation and with increased size of the glia nucleus. The nerve cells in the chronic form of this disorder show an enormous swelling of the whole body with a tendency to disintegration of the cell. In all of the cases one finds the Heubner type of endarteritis in the vessels of the base of the brain without adventitial infiltration and without mesarteritic changes.

In 1904, Alzheimer described three more cases and gave as one of the essentials, disorganization and intertwining of the coats of the vessel wall resulting from a marked proliferation of the cells of the vessel wall. All three of these cases showed microscopically, small areas of softening.

In 1920, Jakob made a study of twenty cases of endarteritis, of which thirteen were pure cases without infiltration, or inflammatory changes, while in seven there was a combination of endarteritis of the small cortical vessels with meningitis or diffuse infiltrative changes similar to those of general paralysis. Jakob found that in almost all cases there was some softening or areas of degeneration, which he believes are the result of arteriosclerotic changes and not essentially specific, agreeing with the concept previously enunciated by Schröder. Jakob agrees essentially with Nissl and Alzheimer that there is a type of syphilitic endarteritis of the small vessels of the cortex characterized by: the absence of lymph sheath infiltration; a marked increase in cells of the vessel wall, with a wiping out of the separation between the

coats of the vessel wall; a considerable degree of proliferation of the protoplasmic elements of the glia cells; and a relatively small amount of diffuse swelling of the ganglion cells and nerve fibers, of irregular distribution over the cortex, with localized increase of the process here and there.

The important point of Jakob's work is that he was unable to discover spirochetes in any of these cases of pure endarteritis. He therefore believes in the theory that the process is of toxic genesis. Jakob further describes one case in which the frontal portion of the brain showed small vessel endarteritis, while in the hind half of the brain a marked infiltrative process was in evidence. In the portion without infiltration he was unable to find spirochetes, whereas in the hind part of the brain where infiltration was considerable, spirochetes were found.

Hauptmann emphasizes this finding of Jakob in building up his theory concerning the pathogenesis of syphilitic and paralytic brain changes. Hauptmann believes that infiltrative appearances speak for the local presence of spirochetes, whereas parenchymatous degeneration and endarteritis suggest a toxic genesis. In other words, from the bacteriologic standpoint, Hauptmann postulates a non-specific process, one that is the result of toxicity of a protein nature, not depending on the presence of spirochetes in the central nervous system.

After reviewing the foregoing, Sioli presents a case of endarteritis in which he found spirochetes. As this has so much importance in relation to the metasymphilis problem, it is presented in some detail.

The patient was born in 1872 and died in 1919. He was picked up on the street confused, disoriented, and grandiose. The pupils were unequal and one did not react to light; the tongue protruded to the left with tremor; the knee jerks were very lively but there was no Babinski nor Oppenheim sign; there was slight speech defect; the heart was increased in size but the heart sounds were clear; the second aortic sound was increased; the Wassermann reaction was positive in the blood and spinal fluid; globulin was present and there was a marked lymphocytosis in the fluid. The man remained euphoric, demented, suggestible, and full of ideas of grandeur; he rapidly deteriorated physically and mentally. He had five mild epileptiform seizures and died in 1919.

At the necropsy there was a fresh hemorrhagic pachymeningitis as well as edema of the pia and chronic leptomeningitis of the convexity. The brain weight was 1,420 gm.

Sections from the central convolution showed increase in size of the vessels, with proliferation of the cells of the vessel walls and an increased number of large, poorly staining nuclei. In many places the layers of the vessel wall could not be distinguished. In several places there was increased vessel formation, which, however, was not general. Rod cells were found sparingly through the cortex. In the adventitial sheath no plasma cells were found. There were no true lymphocytes and only an occasional mast cell, and in several places granular cells with green pigment or basophil-metachromatic content. Likewise in the pia of this region no plasma cells or lymphocytes were found. The ganglion cells were markedly changed with all varieties and degrees of disorder.

The section from the frontal convolution showed a considerable thickening of the pia and a disturbance of the architectonics of the cortex. The endarteritic condition was more marked than in the central convolution. An extraordinarily marked change in the ganglion cells was found. There was

more glia than in the central area with increase in the size of the nuclei and protoplasm and an increased number of glia cells especially about the ganglion cells. Many typical rod cells were found. Around a number of the larger and smaller vessels of the cortex several plasma cells were observed and these were also found in the pia. Further study showed that plasma cells were occasionally present in the frontal and occipital convolutions and in the gyrus rectus.

Spirochetes were found in the basilar artery. They were also found in several places in the central convolution, in the first frontal convolution, and in the gyrus rectus. The basilar artery showed proliferation of the intimal coat, with disorder of the elastica, and in this area colonies of spirochetes were found. These spirochetes were especially fine and thin, the so-called "brown spirochetes."

Aside from these local spirochete colonies, organisms were also found along the entire wall of the basilar artery. In addition, some were found in the coagulated blood within the basilar artery. In the basal pia mater, in the region of the midbrain, several spirochetes were found, and in one small area a large mass of spirochetes was observed. They were also demonstrated in the adventitia both of the pial vessels and of the cortical vessels.

In the central convolution the spirochetes were so few that one could not speak of their relation to the tissue or vessel walls. In the first frontal convolution many spirochetes were present, not diffusely scattered, but in a great many localized areas, so that in almost the entire frontal cortical area spirochetes could be found. They were present in the entire brain tissue but not in such close relation to the vessels as Sioli has described in general paralysis. However, the majority were in the neighborhood of blood vessels, and many were found in the vessel walls.

In the gyrus rectus the spirochetes were less frequent than in the frontal convolution, but in greater number than in the central convolution. No spirochetes were found in the blood coagulum of the vessels of the convexity either in the pia or brain substance. Of the viscera only the tongue and aorta were preserved and in these organs no spirochetes were found.

The case showed histologically, proliferation of the intima of the basilar artery, endarteritis of the small pial and brain vessels with areas of degeneration, heaps of granular cells, areas in the ganglion cells with pericellular effects and marked general glial changes. The endarteritis was present in all portions of the brain studied. In a number of areas there was a pial endarteritis without infiltrating cells. In other places (frontal lobe) the endarteritis was combined with a small degree of plasma cell infiltration, the endarteritis being more marked in the areas where the infiltration occurred.

In general, Sioli states that the case showed the picture described by Nissl of endarteritis and not the type of general paralysis. In summing up the meaning of his findings, namely of spirochetes in a case of endarteritis of the smaller vessels, Sioli states that in three other cases he was unable to find spirochetes and therefore would draw conclusions with a certain amount of reserve. While he is unwilling to make a definite formulation, he still feels that his unique finding robs the toxic theory of much of its potency, as his finding shows that the endarteritis may be associated with the presence of spirochetes.

In regard to the finding of spirochetes in the blood coagulum of the basilar artery within the lumen of the artery itself, he raises the question of whether these were present during life, or were the result of postmortem wandering.

Because of their location in the basilar artery, he is of the opinion that they existed here during life. If this is so, it would lead to the assumption that late brain syphilis is not only the result of spirochetes which have made their way to the brain during the secondary period of the disease, but that they may also reach the brain during any period of the disease, arriving by way of the blood vessels. He maintains that an important question is still to be solved, namely, whether one can make a sharp differentiation between the definite forms of so-called metasyphilis, or whether they are not merely combinations of the same process in different anatomic locations. It seems to him that any hypothesis on this point can well wait until more fundamental work has been accomplished and in this regard he calls attention to the remarkably small amount of work that has been done on this subject.

The paper is illustrated by seventeen photomicrographs showing the histologic and spirochetal findings. It is the first contribution showing the presence of spirochetes in endarteritis syphilitica of the brain, and as such is of the utmost importance in opening a new line of approach to the question of the genesis of this condition. Unfortunately one is not thoroughly convinced that the case is not one of general paralysis complicating endarteritis. Although he states that the histology was not that of general paralysis, the clinical picture, the serologic findings, and the plasma cell infiltration of the frontal areas are suggestive.

, SOLOMON, Boston.

CEREBRAL SYPHILIS AND PSYCHÔSES. L. REDALIÉ, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:230, 1922.

The introduction of lumbar puncture has contributed vary largely to our understanding of the psychoses associated with cerebral syphilis. Koenigstein and Spiegel have recently studied the neuropathologic alterations to be found in patients who did not present clinical evidence of lesions of the central nervous system, but in whom the cerebrospinal fluid pointed to syphilitic involvement. In all of these cases anatomic investigation revealed areas of meningeal infiltration. This observation is of great interest and indicates the great difficulty present in the diagnosis of such conditions. On the other hand there are cases in which the coincidence of a psychosis with syphilis of the nervous system appears to be quite fortuitous.

Herschmann does not hesitate to make a diagnosis of psychogenic depression, hysteria or catatonia in individuals whose spinal fluid gives every evidence of neurosyphilis: regardless of the anatomic evidence of meningeal syphilis these psychoses are to be considered as nonsyphilitic. This is notably true in cases of manic-depressive insanity in which attacks of this psychosis may precede the syphilitic infection. It is impossible in many instances to answer the question as to whether the clinical picture is in any way modified by the presence of cerebral syphilis. Plaut believes that acute and chronic forms of psychoses characterized by hallucinations, ideas of persecution, and anxieties, without grosser evidence of disorientation or impairment of memory are characteristic of syphilis. Marcus divides these cases into types with acute hallucinations, with epileptiform seizures, and with catatonia. Other authors, including Kraepelin, speak of a paranoid, or a schizoprenoid type of cerebral syphilis.

Redalié presents histories of the following interesting cases:

1. A man, born in 1866, constitutionally somewhat depressed, but otherwise quite well. The exact time of his syphilitic infection could not be determined

but was probably not later than 1917. In 1918 his blood Wassermann reaction was positive. In the winter of 1920 to 1921 his vision began to fail, for which he consulted an oculist. A diagnosis of optic atrophy was made and an unfavorable prognosis given. He became depressed, self-accusatory, said that he was poisoning the entire world, that everything he touched became infected; discontinued his work, refused his nourishment, became agitated at night, but did not hallucinate and retained a good memory. Argyll Robertson pupils were present; the patellar reflexes were absent; lumbar puncture done on September 28 revealed positive Noguchi and Wassermann tests, 41 lymphocytes to the cubic millimeter; Wassermann test of the blood negative. He died Oct. 6, 1921. At necropsy a chronic, fibrous, leptomeningitis with lymphocytic infiltration and perivascular infiltration were noted. A syphilitic type of arteritis was observed in a branch of the anterior superior cerebellar artery. The parenchymatous structures were little involved. Pulmonary embolism and general infection were evidently responsible for death.

The interpretation of this case was difficult. One had to consider: (1) a psychogenic depression; (2) hallucinosis of Plaut or the paranoid type of cerebral syphilis; (3) tabetic psychosis; and (4) *confusion délirante* of Marcus. Redalié believes that the emotional trauma in this patient, debilitated by cerebral syphilis, was responsible for the onset of the psychosis. He does not venture to say what course this patient's illness might have taken had he (the patient) not suffered the psychic trauma.

2. A bank employee of good mentality, born in 1885, lost 40,000 francs in speculations in 1917. He became depressed and taciturn. In 1920 he consulted numerous physicians because of gastric crises and vomiting. Albumin was found in his urine. He became fatigued and discouraged and asked to be killed. On admission to the hospital in January, 1921, he seemed indifferent, lost appetite, was negativistic and had to be fed by tube. He was extremely emaciated, the pupils were unequal and irregular but reacted normally. Lumbar puncture showed 14 lymphocytes to the cubic millimeter, a positive Wassermann reaction, Noguchi test faintly positive; there was a strongly positive blood Wassermann reaction. In March an irregular temperature appeared and he died of pneumonia on March 26. Necropsy showed an osteomyelitis of the skull with involvement of the superior longitudinal sinus. The meninges were hyperemic in the frontal area and a few small hemorrhages were noted over the convolutions. There were no findings indicating a general paralysis and no plasma cells could be found. It seems that in this case the organic involvement of the brain was of greater importance than in the preceding one. The preservation of memory and intelligence is to be noted.

3. A woman, born in 1888, at the age of 9 years suffered from convulsions which disappeared after six months. She led the life of a prostitute for ten years, but denied syphilis; was imprisoned on several occasions; drank one to two liters of wine and a good deal of brandy per day; and she used cocaine for a year. The illness for which she was confined to the hospital began in July, 1920, when she became depressed, imagined that no one cared for her, developed ideas of persecution, thought the black hand was after her, and that she was being hypnotized. There was a certain indifference of affect; memory and intelligence seemed preserved. The pupils did not react to light, spinal fluid examination revealed a faintly positive Wassermann reaction, 10 lymphocytes to the cubic millimeter, no increase in meningeal permeability to sodium nitrite, blood Wassermann test negative. A letter written in July, 1921, by

herself indicated that she had recovered entirely and thanked the authorities for having restored her to health from such a serious illness. In this case cocaine, alcohol and syphilis had to be considered as etiologic factors; the type of psychosis added to the difficulty of diagnosis.

4. A married woman, born in 1879, who probably contracted syphilis from her husband, aside from being somewhat nervous, seemed quite well until 1914. In 1915 she was admitted to the hospital because of depression, threats of suicide, visual hallucinations, and delusions of persecution. She was disoriented as to time and place. Later she became euphoric and erotic. The pupils were irregular, reacted poorly to light but well to convergence. The first lumbar puncture gave: positive Noguchi, Lange and Wassermann tests; normal lymphocyte count. A second puncture in May, 1918, showed: 0.5 per cent. albumin; a faintly positive Nonne test; positive Noguchi and Wassermann reactions; no cells. She later became incoherent, was indifferent, laughed without cause, was subject to violent spells of anger without reason, hallucinated, and became stereotyped. A third lumbar puncture in August, 1922, was negative throughout; the Wassermann reaction of the blood was negative. In this patient there was a catatonic state with negativism, indifference, stereotypy, and dissociation of ideas. In 1918 the spinal fluid indicated syphilis; in 1922 it was normal.

5. A woman, born in 1879, addicted to alcohol, whisky, absinthe, and wine, in 1911 developed ideas of persecution, became extremely jealous and later taciturn and negativistic. She had auditory and visual hallucinations; for three years she menaced her husband, her children, and a cousin, with whom she said her husband had intimate relations; accused her husband of defloration of his daughter. She was admitted to the asylum in 1914, at which time she was oriented and seemed to be lucid; answered questions slowly but evasively and was negativistic. There was anisocoria, paresis of pupillary reactions, patellar reflexes greatly diminished. Lumbar puncture showed: a slightly positive Noguchi test, positive Wassermann reaction, no lymphocytes. She was returned to her family in 1914, after which the difficulty again appeared; she was recommitted to the hospital in 1915 because she wanted to put out the eyes of her husband. A second lumbar puncture gave the same results as the first. The third puncture fluid was negative throughout, and the Wassermann reaction of the blood was negative.

Redalié calls attention to the fact that in all of these cases syphilis was undoubtedly present and that the clinical picture was extremely varied. Further than this he permits the reader to draw his own conclusions.

WOLTMAN, Rochester, Minn.

STUDIES OF CEREBROSPINAL FLUID AND BLOOD OF SYPHILITIC AND NORMAL PERSONS, WITH SPECIAL REFERENCE TO THE IMMUNITY REACTIONS AND THE COLLOIDAL GOLD TEST ON THE ORIGINAL AND UNFILTERED FLUIDS AND SERUMS.
CHARLES E. NIXON and KOICHI NAITO, Arch. Int. Med. **30**:182 (Aug.) 1922.

The first part of this extensive research is concerned primarily with the nature of the substance in the serum responsible for a positive Wassermann reaction. The globulin and albumin were separated from the serum: globulin was precipitated with ammonium sulphate solution and purified by dialysis; the albumin fraction was filtered and dialyzed in the same way. The Wassermann reaction on albumin from syphilitic serums was negative in all instances;

whereas the reaction on globulin was positive in all instances with a marked tendency to an increase in the positivity. The globulin from normal serums gave a positive reaction in 72 per cent. of twenty-one cases. The authors conclude that the globulin fraction in syphilitic serums contained the active substance in the Wassermann reaction. Experiments on the filtrability of globulin in syphilitic serum by the ultrafilter showed that the filtrability was less than normal serum. Three factors are advanced to explain the last observation: (1) the possible ease of adsorption of the active globulin of the ultrafilter as compared with normal globulin; (2) the possible greater size of the particles of active globulin as compared with normal globulin, and (3) the possible instability of the active globulin as compared with normal globulin.

The second part of this research deals with the nature of the colloidal gold test. Globulin prepared from normal human blood serum gave distinct curves, the reduction usually occurring in Zone I; curves from globulin from syphilitic serums were somewhat more marked. The albumin fraction from normal and syphilitic serums may cause a reduction, the curve occurring in the moderate dilutions. Dialyzed syphilitic and non-syphilitic serums gave curves tending to be irregular. Colloidal gold reactions on the original and ultrafiltered serums gave fairly similar curves with a tendency to a greater difference between the zones of reduction in the syphilitic cases. The globulin:albumin ratio was found not to be a determining factor in the gold curve. Ultrafiltration of the spinal fluid was found to lessen the intensity of the curve and also to change the zone of reduction. Addition of salt solution tended to increase the intensity of the curve. Albumin tended to diminish the curve and change the zone of reduction.

The authors conclude that there are present in the cerebrospinal fluid and blood certain substances which act in a protective way against the coagulation of colloidal gold by an electrolyte; that both precipitating and protecting substances are present in pathologic fluid and the curves in Zones I, II, and III are due to varying amounts and proportions of the precipitating and protective substance; that the protective substance is decreased by ultrafiltration to a greater degree than the precipitating substance; that the precipitating and protecting powers are modified by the state of the protein; and that the salt solution used in the test partially neutralizes the protective action.

VONDERAHE, Philadelphia.

THE VIBRATING SENSATION IN DISEASES OF THE NERVOUS SYSTEM. R. T. WILLIAMSON, *Am. J. M. Sc.* **164**:715 (Nov.) 1922.

This article is a report of the use of a large vibrating tuning fork in the study of nervous diseases extending over a period of seventeen years. Many obscure forms of early organic disease of the nervous system are clearly differentiated and, in the author's estimation, the loss of the vibratory sense takes the same place in the sensory system as the Babinski sign in the organic changes of the pyramidal tracts.

Normally, the vibrating sensation is felt when the foot of the vibrating tuning fork is placed over the bones of the limbs, on the nails, on the iliac spines, sacrum, vertebral spines and bones of the thorax. It is particularly well felt over the sternum. On the bones of the skull, the vibrations are not felt, but are heard. On the anterior abdominal surface, at the level of the umbilicus, it is distinctly felt under normal conditions. It is also well per-

ceived between the umbilicus and the thorax. Below the umbilicus it may be felt only when firm pressure is used, or, in cases of very stout persons, it may not be felt at all.

Loss of the vibratory sense at any of the above points distinctly indicates organic changes in the sensory nervous system. When associated with other symptoms it may be considered as a positive sign of an organic lesion, and it may be the earliest sign of such a process.

A study of spinal lesions has shown that the nerve fibers conducting this sensation apparently do not decussate in the spinal cord. They are found to ascend in the posterior columns. In cerebral disease the vibrating sensation is lost along with other sensations. Its return is coincident with the return of other forms of sensation. It has no known cortical center.

The loss of the vibrating sensation may be present in the earliest stage of the disease when other symptoms and signs are very few or slight; such as in the earliest stages of a peripheral neuritis, tabes dorsalis or posterolateral sclerosis. It is one of the first indications of sensory affections in many lesions of the spinal cord, and later, loss of tactile, pain and temperature sensations may be found. It is a very delicate test and should be used routinely. In spinal anesthesia the vibrating sensation is often the first to be affected and the last to recover; while in cerebral anesthesia the vibrating sensation may be the least affected, and if affected it may recover before tactile anesthesia. Such motor diseases as anterior poliomyelitis or amyotrophic lateral sclerosis, may be excluded if the vibrating sensation is found to be lost. A paraplegia due to organic disease may be easily differentiated from a paraplegia due to hysteria, functional affections or malingering.

The loss of the vibrating sensation on the abdomen may be of service in indicating the upper limit of a spinal lesion in traumatic and other spinal affections when other forms of sensation are not affected.

Vibrating sensation is usually lost in the following diseases: Multiple peripheral neuritis due to alcohol, arsenic, diabetes, etc.; acute disseminated myelitis; acute myelitis; combined posterolateral spinal degeneration or sclerosis; tabes dorsalis; syphilitic spastic spinal paralysis; compression myelitis due to caries; spinal meningeal tumor. It may be a means of determining the level of localization in spinal cord tumors or injuries and in detecting the early presence of pernicious anemia. The diseases in which the vibrating sensation is never lost are: anterior poliomyelitis (acute, subacute and chronic); amyotrophic lateral sclerosis; sciatica; primary brachial neuritis; lesions of a single peripheral nerve; hematomyelia; syringomyelia; cerebral diseases in which other forms of sensation are retained; hysteria; functional affections, neurosis; neurasthenia, and malingering.

The paper presents in detail the finer points of differentiation between these diseases.

TEMPLE FAY, Philadelphia.

TUBERCLE OF THE TEGMENTUM PONTIS—A CONTRIBUTION TO THE SYMPTOMATOLOGY OF PONTINE LESIONS—PICK'S VISIONS. H. W. STENVERS, *Schweiz. Arch. f. Neurol. u. Psychiat.* **11**:221, 1922.

The patient was a shoemaker, forty years of age, who had often had attacks of coughing during childhood and at thirteen years of age had an hemoptysis followed by a five months' rest cure. Three and one-half months previous to admission he had attacks several times a day in which he felt compelled to

swallow very rapidly over a period of about three minutes. These attacks began suddenly and were accompanied by a sour taste in the throat. After nine weeks this symptom disappeared. Five weeks previously he developed a sensation of pepper in his mouth and nose, suffered from attacks of dizziness, and had difficulty in speaking due to a "swollen tongue." Three weeks later he was unable to swallow when he tried to do so voluntarily, but did well involuntarily: paresthesia in both arms and diplopia soon appeared; there was morning nausea and vomiting. The dizziness interfered with his walking, especially so when looking at a moving object. There was no headache at any time. Urination and defecation were normal; erections were maintained, however ejaculation had been absent for eight months.

At the time of the examination in April, 1921, there was dysarthria, a normal fundus, horizontal nystagmus in looking toward the right, left or upward, diplopia on looking toward the left due to weakness in the field of the left external rectus, normal corneal reflexes, slight hypalgesia over the tongue, palate, and lips, slight weakness of the left facial, inability to hear watch on the left, past pointing laterad with either hand, atrophy of the left half of the tongue with deviation toward the right on protrusion, nasal speech, hypotonia of the right arm, general cutaneous hyperpigmentations, normal reflexes throughout, some uncertainty in gait, good muscular coordination, seven lymphocytes and a faintly positive Nonne in the spinal fluid.

While in the hospital the patient was restless, sang, and complained of vertigo. He later hallucinated, was often somnolent with attacks of arrested respiration, and complained greatly of fatigue. Early in May he said that cold water was being thrown on his face, that he heard someone rapping on the bed, that he saw remarkable things going on in the adjoining room, that people were sitting on the radiator and were jumping through the walls, that the walls of the room were bent forward and moved upward. These special hallucinations were prominent and persistent. The patient died May 8 of respiratory failure.

The delirious condition at first suggested a diffuse process; however, the peculiar visual hallucinations, first described by Pick, in which he emphasized the influence of cerebellar, vestibular, and ophthalmostatic disturbances on these, suggested a lesion in the region of the fourth ventricle, particularly of the floor.

Necropsy revealed a tubercle in the tegmentum of the pons and the medial and dorsal portion of the medulla. The upper end of this tubercle lay just caudad to the abducens nucleus, and the lower end was in the plane passing through the inferior extremity of the left olive. The posterior longitudinal fasciculus was involved.

Stenvers calls attention to the early disappearance of ejaculation in the presence of normal erections. While this dissociation has been looked on as pathognomonic of epiconus lesions, it can evidently be dependent on disturbances higher up. The occurrence of ejaculation in hemorrhage of the fourth ventricle is well known. It is likely, therefore, that ejaculation proceeds from the medulla or above. According to Oppenheim, erection probably has its center in the second sacral segment while the center of ejaculation lies below this point. The writer's observation would localize this reflex tract as passing through the district lying between the abducens and the hypoglossal nuclei.

The difficulty of orientation in space in the presence of optical stimulation by moving objects is interesting. Probably the lesion of the posterior longitudinal fasciculus was responsible for this. Pick attempts to interpret the seeing

of visions through a wall as a result of diplopia. In this patient such hallucinations were present in the absence of diplopia, although there was, to be sure, a paresis of the ocular muscles. Stenvers believes that Pick's visions are of some diagnostic importance, indicating a lesion in a circumscribed portion of the floor of the fourth ventricle.

WOLTMAN, Rochester, Minn.

AFTER-CARE OF INFANTILE PARALYSIS CASES OF THE 1916
EPIDEMIC IN BROOKLYN: A Detailed Report of About Three
Hundred Cases Followed Through Five Years. H. G. DUNHAM; with
an Introduction by THOMAS J. RILEY, J. A. M. A. 80:224 (Jan. 27) 1923.

This is a report of the repeated examination of 297 patients chosen from the thousands stricken with infantile paralysis in Brooklyn in the summer of 1916, and followed through five years. It is probable that a large proportion of the population at sometime in their life history has been afflicted by the abortive or nonparalytic form, and it is conceivable that a certain number of obscure conditions of the central nervous system may have been initiated by adhesions and obstructions resulting from such an attack, unnoticed at the time of onset because no clinical signs and symptoms were present.

Of special significance is the observation that only a small group of patients previously operated on for tonsil and adenoid conditions contracted the disease, and in the patients operated on the percentage of recoveries was very much higher than in those who had not been subjected to operation.

It was determined that persons with the disease were a source of active infection at least eight days after the acute onset, and there was little evidence that the disease was contracted from a person ill more than two weeks. It is thought that the virus is carried to the different parts of the body by the lymphatic system, and, contrary to the older studies, the disease at present must be considered a general and not a local infection, but showing a tendency to localize in the nerve and lymphoid system.

The number of cells in the cerebrospinal fluid is increased to from 20 to 100, and in some cases even as high as 500 per cubic millimeter. Polymorphonuclear cells predominate in the earlier stages of the disease, preceding the paralytic period. After paralysis is established the mononuclear lymphocyte predominates in numbers, ranging from 75 to 100 per cent.

In this group only fourteen families had more than one child ill with this disease at the same time.

Particularly during an epidemic of infantile paralysis of any size, children with obscure indispositions should be kept at rest in bed for several days, for if paralysis supervenes, it usually appears within a very short time after the initial upset, the average for this series being three days after the first manifestation of illness.

In a general way, the younger the child, the nearer does restoration of function approximate the normal status. Hydrotherapy was very generally advised for all these patients, and by the majority was faithfully carried out. When possible, the child was taught to exercise the affected muscles while in warm water, which was an advantage, the extremities being lighter in water than in the air.

In this series, the back and abdomen with both lower extremities was the combination of involvement by far most frequently found. The more severely involved upper extremities did not respond to treatment as rapidly as the equally involved lower extremities.

NIXON, San Francisco.

THE MYOCLONIAS. H. ROGER, *Ann. de méd.* **12**:150 (Aug.) 1922.

This is a critical review of the subject of the myoclonias, embracing its historic and clinical aspects as well as its pathogeny and treatment. Concerning the pathogeny, the theory of a myopathic origin rests only on necropsies showing alterations in muscles. Roger is certain such muscle changes are secondary and not primitive. The neuritic origin, defended by Manquat and Grassat, was based on the coexistence of paramyoclonus and neurofibromatosis with abolition of the reflexes in several cases. But Vanlair experimenting in a dog with myoclonus did not find the latter suppressed by curare, a poison of nerve terminals. At the present time, therefore, the discussion limits itself to the spinal theory as opposed to the cerebral.

Friedreich related paramyoclonus to an exaggerated excitability of the spinal anterior horns. Points of evidence for this conception given by Friedreich were: the symmetry of the movements; the great influence of reflex cutaneous excitations; and the abolition of the movements by voluntary movements. The presence of fibrillary contractions in both the poliomyelitides and the myoclonias is another argument. The author cites two cases, both with myoclonus localized in the legs. This localization, with the coexistence of sphincteric troubles, and bilateral positive Babinski sign, are in favor of a spinal origin of the movements.

The chief sponsor for the cerebral theory is Raymond. He incriminates cerebral lesions and points out the association of epileptic crises with certain myoclonic syndromes, and also cites the findings of several necropsies: a sclerosis in the rolandic zone in an epileptic case with myoclonus (Jacquin and Marchand), and lesions, predominantly cortical, in a case of acute paramyoclonus (Murri).

There is a tendency to incriminate the mesencephalon and in particular the corpus striatum by reason of the known localization in these centers of a great number of automatic movements. Roger writes, "Although the majority of the present facts in regard to encephalitic myoclonias are in favor of the striate origin, the spinal hypothesis cannot as yet be rejected. The number of necropsies with complete histopathology is not sufficient to settle the problem." Roger shows a disposition, to ask, with Hunt, if two localizations are not possible. Hunt has suggested that rhythmic myoclonias relate to a disordered infrastriate mechanism, and that non-rhythmic myoclonias (paramyoclonus) relate to lesions of the cord or medulla.

DAVIS, New York.

THE INFLUENCE OF THE NERVOUS SYSTEM ON THE DEVELOPMENT AND REGENERATION OF MUSCLES AND INTEGUMENT IN INSECTS. STEFEN KOPEC, *J. Exper. Zool.* **37**:15, 1923.

The fifth abdominal ganglions of caterpillars of *Lymantria dispar* L., were removed after the second moult. The animals developed normally and after three to five weeks they were preserved and studied in sections. The muscles of the caterpillars did not undergo either atrophy or degeneration after removal of their corresponding ganglions. All three thoracic nerve ganglions were removed from caterpillars a few days before pupation and the behavior of the muscles was studied in the emerging moths. In the moths muscles were found only when the larval ganglion of the corresponding segment had been left intact. On the contrary, the adipose cells of the caterpillars underwent metamorphosis notwithstanding the absence of the corresponding ganglions. The hypertrophic growth of the tracheas and of the intestine in imaginal seg-

ments deprived of muscles, points to mechanical correlations regulating the normal size of the organs. In general the nerve ganglions do not influence the normal development of the hypodermis of the full-grown insect, nor that of its chitinous integument together with the wings, legs, and feelers. The fourth abdominal, segmental extremities together with their corresponding ganglions and nerves were removed from caterpillars after their last moult. The abdominal legs regenerated quite independently of the nervous system as well in regard to their macroscopic as to their histologic structure.

WYMAN, Boston.

A CONTRIBUTION TO THE PSYCHOPATHOLOGIC AND LOCALIZING VALUE OF PARALLELISM BETWEEN PSYCHIC AND MOTOR ACTIVITY. H. STECK, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:208, 1922.

The writer begins his article with the conclusion that in normal as well as in pathologic conditions there exists a parallelism between motor and psychic activity. This is seen normally in the fact that walking aids thinking. In two cases of epilepsy observed by Steck, verbal perseveration went hand in hand with motor perseveration. In the catatonic stupors this parallelism is evident; all initiative is lacking in both motor and psychic fields, although it is well known that the patient is able to register his sensations. The most striking condition in which this phenomenon is observed is in post-encephalitic parkinsonian states. In all of these cases automatic movements were accompanied by an apathy and a loss of interest apparent to both patient and relative. As far as the investigations of this condition give us an insight into it, there is seen poverty and slowness of thought and action. It is worthy of note that in some of these patients a notable lack of energy constituted a character weakness, present even before the onset of the encephalitis; it is conceivable that this poverty of activity may be bound to a constitutional weakness of the striate apparatus, which thus would constitute a locus of lessened resistance susceptible to exogenous intoxicants. In contrast to these akinetic forms, the hyperkinesias, such as the choreas, athetoses, and tics, are accompanied by lability of effect, irritability, hypervigility, and hypotenacity of attention.

Dupré spoke of the correlation of oligophrenia with motor debility; Joffroy coined the term "myopsychies" to designate this sympathy of motor and psychic functions. Motor activity is thus a mirror picture of the psychic processes, and the reverse. It is true that there are exceptions to this rule, just as we find exceptions in the mixed states of manic-depressive insanity. Thus, irritability in a case of Parkinson's disease does not necessarily contradict this thesis. Necropsy findings in these encephalitics show involvement of the basal ganglions, particularly of the corpus striatum. In some cases there were also marked lesions of the frontal lobes.

Steck concludes that one may expect to find involvement of the frontostriate system whenever a pathologic psychomotor parallelism can be demonstrated.

WOLTMAN, Rochester, Minn.

COMPARATIVE CLINICAL OBSERVATIONS ON THE INVOLVEMENT OF THE NERVOUS SYSTEM IN VARIOUS PHASES OF SYPHILIS. JOHN H. STOKES and ALBERT R. McFARLAND, *Am. J. Syphilis* **6**:169 (April) 1922.

The authors studied the results of spinal fluid examination in four groups of syphilitic patients: early and late secondary syphilis, one group from the

Mayo Clinic, Section of Internal Medicine, and another group from a consultant practice. They conclude that treatment of neurosyphilis in the first two years gives a marked response; that a rise in a cell count of the cerebrospinal fluid is the earliest evidence of a meningeal reaction; increased globulin, and finally positive Wassermann reaction follow; while symptoms and signs are regarded as late rather than early signs of involvement of the central nervous system. Spinal puncture should not be made, in their opinion, until one or two injections of arsphenamin have been given; but the routine examination of spinal fluid is advised in all cases of early syphilis. Neurosyphilitic involvement takes place early; if the spinal fluid is normal during the early secondary period there is a distinct immunity toward subsequent involvement, 40 to 50 per cent. of such patients having normal spinal fluid at all stages. The vital period of the disease which offers the most favorable opportunity for preventing involvement of the central nervous system is in the first two years. In a group of 16 to 18 per cent., which includes both early untreated and late syphilis, both blood and spinal fluid appear normal. A proportion of patients varying from 5 to 8 per cent. in early syphilis, and from 14 to 41 per cent. in late syphilis, will present negative blood reactions but abnormal spinal fluids. The authors emphasize the importance of spinal fluid examination. The Wassermann reaction in syphilitics was found to be 50 per cent. positive on blood as compared with 60 to 70 per cent. positive on spinal fluid.

VONDERAHE, Philadelphia.

A NEW CONTRIBUTION TO THE STUDY OF THYMIC DEATH.

A. PULAWSKI, *Rev. de méd.* **39**:495 (Aug.-Sept.) 1922.

A young woman subject to rather prolonged and severe hysterical attacks was first seen several hours after the onset of one of them. She was unconscious, cyanotic, showed a feeble pulse, had irregular respiration, not of the true Cheyne-Stokes type, but "recalling rather the 'suffocation' of hysterics." Although she did not have myosis, but because of a faint suspicion that she had taken morphin with suicidal intent, she was treated actively by gastric lavage, injections of atropin, camphor, caffein, and artificial respiration. Later under the application of faradism, she temporarily regained consciousness and addressed two of the persons near her. Then she relapsed into apparent unconsciousness, and only a few hours later, while still in a profound and calm slumber, without any convulsion, having a respiration which had become regular, she died.

In his discussion of the question, "Can death be due to an hysterical attack?" the author brings forward the necropsy findings in the case. The patient presented a status thymico-lymphaticus. The thymus weighed 47 gm. The author believes that the death was in reality a "thymic death" and recalls that a moderately large group of persons who display the thymico-lymphatic condition are menaced either casually or after an insignificant accident, by sudden death.

Contrary to the former experience of the author, he found a large hyperplastic suprarenal medulla present. Another unusual feature was an outstanding "hypertrophy of the brain," so that it appeared that the brain lacked sufficient space. Such a feature has itself been claimed as a cause of sudden death. (Neusser, Hornowski, Srzywo-Dombrowski.)

DAVIS, New York.

SPASMODIC FORCED RESPIRATION AS A SEQUEL OF EPIDEMIC ENCEPHALITIS. IRVING H. PARDEE, J. A. M. A. **80**:178 (Jan. 20) 1923.

The author describes certain disturbances of the respiratory mechanism that have come under his observation as a sequel to epidemic encephalitis, which have resulted clinically in disorders of breathing of a very bizarre nature. He has noted the type described by Aronson in which the respiratory rate is over forty and the effort loud and labored, suggesting the panting of a dog. In five other cases there was a type of disturbance in which intervals of normal breathing were interspersed with occasional outbursts of abnormal respiration. Without any premonitory feelings other than, perhaps, slight dizziness, the patients complain that they feel compelled to take a deeper breath; that something is compressing the lower chest which interferes with adequate depth of breathing. The breathing, therefore, suddenly becomes deeper and more rapid, usually between forty and forty-five a minute. The effort is marked, inspiration being voluntarily long and deep, with all accessory muscles called into play. Expiration is also voluntary and labored, frequently ending with a noticeable grunt. During these spasmodic paroxysms, the mental attitude is obviously one of distress and anxiety, suggesting that which is sometimes seen in vagal attacks, and is concentrated on the difficult breathing. Conversation is avoided, and, if the breathing is voluntarily interrupted, the succeeding respirations appear to be more violent, as though to compensate.

Nixon, San Francisco.

STUDIES ON THE RETINA. PHOTOMECHANICAL RESPONSES IN THE RETINA OF *EREMIAS ARGUS*. S. R. DETWILER, J. Exper. Zool. **37**:89, 1923.

Specimens of *Eremias argus*, a diurnal lizard common in the vicinity of Peking, China, were kept under varying conditions of light or darkness following which the eyes were prepared for histologic examination. Studies of the retina brought out the following facts: The retina of *Eremias argus* possesses single and double cones and is entirely devoid of rods, resembling in this respect that of other diurnal saurians. It is also characterized by the possession of a slightly developed fovea, situated in the center of a prominent "area." It also contains a prominently developed pecten of conical shape which is highly vascular and deeply pigmented. The foveal cones are much more numerous and much more slender than those in the extrafoveal region. They exhibit, however, all of the characteristics of typical cones in possessing conical outer segments, oil drops and paraboloids. Illumination of the eye by diffuse daylight brings about a slight contraction of the cones and a migration of the pigment. The extent of the cone contraction averages 2.3 microns in the foveal region and 2.9 microns in the extrafoveal region. The pigment migration averages 4.1 microns at the fovea and 3.4 microns in the extrafoveal region. The photomechanical responses observed in this eye, as well as those obtained from a study of the eyes of the turtle, *Chrysemys picta*, and the lizard, *Sceloporus undulatus*, have a distinct bearing on the question of the functional significance of pigment migration and changes in the visual cells in light and dark adaptation.

WYMAN, Boston.

ASTHENIA AND MANIA FROM ANTITYPHOID VACCINATION. R. BENON, Rev. de méd. **39**:585 (Oct.-Nov.) 1922.

The unusualness of the viewpoint gives this report interest. The author reports that an antityphoid vaccination brought about in a patient marked

asthenic symptoms; then a manic phenomenon and for over five years he has continued in a psychotic condition, difficult of diagnosis with alternate periods of excitement and of depression, and at times great inactivity and even stereotypy.

The author believes that asthenia (amyosthenia and anideation) so common, if not constant, after infectious states, can be instituted also by anti-typhoid vaccination. These are exceptional cases which are in relation with individual predispositions. He does not regard them as contraindications for such vaccination.

DAVIS, New York.

THE VALUE OF NEUROLOGICAL EXAMINATION IN SYPHILIS.

RANDAL HOYT, *Am. J. Syphilis* **6**:273 (April) 1922.

Hoyt calls attention to the observation that many early cases of neurosyphilis can be cured, and suggests that a very complete neurological examination be made and repeated at intervals. Cases studied by Hoyt often showed only slight neurological manifestations such as hypesthesia of feet and ankles and slight ocular affections, these being manifested as isolated symptoms and of such a character as to be overlooked unless examination is very thorough.

VONDERAHE, Philadelphia.

DYSTHYROIDISM, ANAPHYLAXIS, AND EPILEPSY. By V. M.

BUSCAINO, *Schweiz. Arch. f. Neurol. u. Psychiat.* **11**:261, 1922.

In 1915 Buscaino stated his belief that idiopathic epileptiform seizures represent an anaphylactic crisis which is caused by penetration into the blood stream of abnormal thyroid products. These abnormal proteins appear in the form of octahedral crystals which may be demonstrated in the thyroid by staining frozen sections, fixed in ten per cent. formaldehyd solution, U. S. P., in a saturated solution of Nile blue sulphate for eighteen hours; differentiate in a 2 to 3 per cent. solution of acetic acid; wash in distilled water, and embed in glycerin. These crystals are insoluble in water, alcohol, acetone, sulphuric ether, mineral acids, acetic acid, and alcohol; they do not stain with Sudan III, are not inorganic, and do not consist of fats or carbohydrates. The xanthoproteic reactions and the reactions of Millon, Liebermann-Würster, and Axenfeld are positive. The crystals are, therefore, protein. They are usually found in the colloidal material, sometimes singly, sometimes in great numbers.

The crystals are definitely related to epilepsy. In all 396 thyroids were investigated. The thyroids of epileptics or other patients having epileptiform seizures revealed the crystals in 71 per cent.; in the thyroids of nonepileptics they were found in 14 per cent. In addition to cases of epilepsy, they may be found in progressive paralysis, arteriosclerotic dementia, idiocy and mental deficiency, brain tumors, uremia, alcoholism, and were found in one case of cysticercus cyst. These abnormal proteins enter the blood stream from the thyroid and produce an anaphylactic crisis against which the body reacts by the formation of specific reagents.

Crile has removed one adrenal and three fourths of the thyroid gland and sectioned the cervical sympathetic in 11 cases of epilepsy with a good effect on the severity of the attacks and the length of the interparoxysmal period. The writer draws a therapeutic conclusion, saying that removal of the entire

thyroid for the purpose of eliminating the source of these abnormal proteins, and the subsequent administration of thyroid preparation should produce a beneficial effect on these convulsions.

WOLTMAN, Rochester, Minn.

FURTHER FACTS REGARDING THE INFLUENCE OF FEEDING THE
ANTERIOR LOBE OF HYPOPHYSIS ON THE RATE OF GROWTH
AND THE SIZE OF AMBYSTOMA TIGRINUM. E. UHLENHUTH,
J. Exper. Zool. **37**:101, 1923.

Metamorphosed salamanders of the species *Ambystoma tigrinum* were fed on earthworms, on beef liver and on anterior lobe of cattle hypophysis. Beef liver causes a rate of growth higher than that produced by earthworms and as high as that produced by anterior lobe. The characteristic peculiarity of anterior lobe when fed to salamanders is to maintain growth for a longer period and beyond a larger size than beef liver. Anterior lobe, among the food substances tested so far, produces the largest animals. Animals fed anterior lobe may reach a size 25.4 per cent. in excess of the size of the largest known normal animal of this species, while the largest liver-fed animal exceeds the normal maximum size of the species by only 5 per cent. Since the age limit for growth was reached in none of the animals which formed the material for this report, it seems that the cessation of growth was determined by the size up to which a given substance can make the animals grow. The effects of the anterior lobe feeding upon the final size of the salamanders are not due to quantitative differences in the food and age of the animals, but to a qualitative difference existing between anterior lobe and other food substances. For this reason the assumption that anterior lobe substance does produce real gigantism in certain salamanders, is apparently even more justified today than it was previously.

WYMAN, Boston.

SOME OCULAR PHASES OF LITTLE'S DISEASE (CONGENITAL
SPASTIC RIGIDITY OF THE LIMBS). WILLIAM C. POSEY, J. A.
M. A. **80**:80 (Jan. 13) 1923.

The author reports three cases of Little's disease with ocular symptoms, and discusses the types of ocular involvement found in these patients. Strabismus is the most constant and nystagmus occurs next in relative frequency varying greatly in degree and kind. Paralysis of the extra-ocular muscles and optic atrophy are rare.

NIXON, San Francisco.

ANTITYPHOID VACCINATION AND GENERALIZED SCLERODERMA.
NICHOLAS and GATÉ, Bull. méd. **36**:843 (Oct.) 1922.

A diffuse scleroderma, almost completely generalized, developed rather acutely in a man aged 44 following a series of antityphoid vaccine injections. (Two previous vaccine series.) The patient presented no signs of tuberculous infection and no pathology was discoverable as a possible cause of the scleroderma. "The relation of cause and effect between the vaccination and the scleroderma appears probable." A single case of this sort merits publication because of its possible bearing on a procedure, namely antityphoid vaccination, the incontestable usefulness of which received full proof during the recent war.

DAVIS, New York.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, March 15, 1923.

F. K. HALLOCK, M.D., *President, in the Chair*

TWO CASES OF CEREBRAL HEMORRHAGE IN YOUNG ADULTS.

DR. BENJAMIN T. BURLEY.

The following two cases were under my observation simultaneously in April, 1922, at the Worcester City Hospital:

CASE 1.—A man, aged 18, member of a healthy family, who had been generally well except that his left eye had been injured and enucleated the year before entrance to the hospital. April 18, 1922, he noticed, while at his work as machinist, that he did not feel as well as usual and developed a severe headache. At his father's suggestion, he started to return home but was seen to stagger and fall while on his way. His father followed and took him home in an automobile. His physician was called and twice administered $\frac{1}{4}$ grain (0.015 gm.) of morphin without much effect on the headache. I saw him in consultation the next morning and ordered his removal to the Worcester City Hospital. When examined, the patient was writhing in pain from headache, particularly of the right side. He complained also of photophobia and blurred vision. The right pupil was somewhat dilated but it reacted promptly; the left eye was missing. The knee jerks were lively, the left greater than the right. There was a tendency to ankle clonus on the left and positive Babinski and Oppenheim reflexes on the left. There was moderate *tâche cérébrale*; anesthesia was absent except for a complete astereognosis of the left hand; coördination of movements of the left arm and leg was imperfect; and there was no nystagmus nor muscular tremor. The temperature was 98.6; pulse, 56; blood pressure, 130 systolic, 70 diastolic. The spinal fluid was sanguinolent, under moderate pressure, and contained increased albumin and globulin; the Wassermann reaction was negative. The white blood cell count was 8,800.

Increased intracranial pressure was indicated by the rapid development of choked disk in the right eye and the headache became so unbearable that it was decided to perform a right exploratory craniectomy. An area $3\frac{1}{2}$ inches in diameter over the parietal and upper temporal lobe was exposed, and disclosed a non-pulsating brain with somewhat flattened convolutions and resistance in the area of the posterior central gyrus. A blunt trocar inserted in this area yielded a dark clot from the depth of $\frac{5}{8}$ inch. Ventricular tapping also evacuated old blood clots with some hemorrhagic fluid. After a sub-temporal decompression with drainage, the skull-flap and skin-flap were replaced.

Absolute relief of the headache followed the operation, vision returned on the following day so that flowers could be distinguished across the room and the patient made a slow but practically uninterrupted recovery. After two or three weeks the astereognosis and spasticity of the left side had practically disappeared. The neuroretinitis of the right eye subsided markedly and the patient returned home after one month. He later returned to work and has since remained free from cranial symptoms.

CASE 2.—A single woman, aged 31, an office clerk, with a family history devoid of importance, had a fibroid removed from the uterus a year and a half before entrance to the hospital and made a rather slow recovery because of a tendency to exhaustion. Although lacking somewhat of her usual animation in recent months, she had no significant symptoms until one week before entering the hospital. One morning as she stooped over she was seized with a terrific headache over the right eye. This pain lasted for from two to three hours and was accompanied by nausea and vomiting. She received "osteopathic" treatment before entrance to the hospital. Four days after the first attack, the right eyelid began to droop; two days later she had another sudden terrific headache lasting about twelve hours. This attack was preceded by a convulsion, and was succeeded by vomiting. She was taken to the City Hospital April 20, 1922.

Examination revealed a well developed and well nourished young woman, with paralysis of the third cranial nerve on the right. There was no other cranial nerve involvement, and no sensory disturbance; a moderate *tâche cérébrale* was present but no incoördination and no vasomotor disturbance. Headache was severe over the right eye. The pulse was 50; temperature 98.1; white blood cell count 16,000; blood pressure, 122 systolic; 84 diastolic. The fundi at this time were normal. Lumbar puncture yielded a fluid containing old blood; albumin and globulin were three plus; smears disclosed no organisms; and cultures produced no growth. The colloidal gold reaction was 1,111,231,100. After four days, the headache still persisted. Lumbar puncture was again done and yielded blood-stained fluid under moderate pressure. There was some relief from this puncture; the pulse rose from 50 to 60; and the white cell count was now 14,200. After being in the hospital one week, optic neuritis began to appear in the right eye and, a little later, in the left.

April 30, ten days after entrance to the hospital, the patient again had sudden terrific pain over the right eye, with a convulsion lasting eight minutes. Two hours later lumbar puncture was done and 25 c.c. of bright, fresh, deeply blood stained fluid was removed with some relief of symptoms.

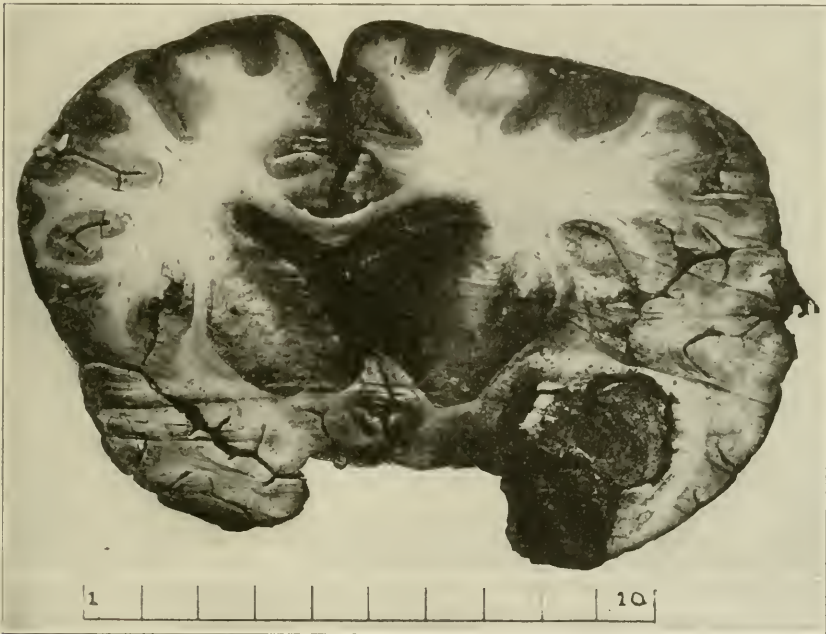
May 6, 1922, the patient was seen in consultation by Dr. E. W. Taylor. She then had definite edema of the disks of both eyes, four diopters of swelling in the right and two in the left, with some hemorrhagic areas that were more marked on the right. Lumbar puncture was again done, the fluid at this time being less bloodtinged than at the time of the convulsion. The presence of choked disks and failure to relieve the headache led to the decision to do a decompressive craniectomy on May 12. An opening two and a half by two inches over the right temporal lobe exposed a brain with a thin layer of blood clot and edema, non-pulsating and with flattened convolutions. Decompression only was attempted. The patient experienced some relief of her headache from the operation but her general condition was not improved. May 17, she had a series of generalized convulsions in one of which she died.

Necropsy disclosed a hemorrhage extending from the tip of the right temporal lobe backward and inward, communicating with the lateral ventricle and filling the ventricular system throughout. The brain was hardened in formaldehyd solution and sent to Dr. Taylor for pathologic examination.

PATHOLOGIC EXAMINATION BY DR. E. W. TAYLOR

Macroscopic Appearances.—There were present: Extensive subpial hemorrhage on the mesial and inferior aspects of the right temporal lobe, also in

the region of the cisterna magna of the oblongata, especially on the left side, extending over the cerebellum in the median line and also somewhat laterad; no hemorrhage in the interpeduncular space; considerable collection of blood in the cerebellopontine angle, especially on the left side; the third nerve on the right more or less compressed by hemorrhage. On section of the brain through the anterior portion of the temporal lobes, a large hemorrhage about 3.5 x 2.5 cm. in size was found occupying the tip of the lobe on the right, reaching the surface inferiorly and mesially. This hemorrhage had ruptured into the descending horn of the right lateral ventricle, which was filled with blood. At this point there was much less blood in the left ventricle; sections posteriorly showed both lateral ventricles and the third ventricle completely



Section through the anterior part of the temporal lobes in Case 2.

filled with blood; frontal section through the oblongata and cerebellum showed the fourth ventricle to be completely filled also. The course of the hemorrhage was evidently from the original focus in the right temporal lobe through the descending horn of the right lateral ventricle and thence through the other ventricles, appearing finally in the cisterna magna, and thence extending somewhat over the cerebellum and in the cerebellopontine angles.

Microscopic examination shows the brain tissue destroyed and invaded by hemorrhage, without evidence of tumor. Sections made through the temporal lobe in the neighborhood of the primary hemorrhage and through the region of the third nerve nucleus do not show characteristic lesions of encephalitis and although this diagnosis cannot therefore be substantiated, it is possible that a more complete examination of other portions of the brain might lead to a different conclusion.

DISCUSSION

DR. B. T. BURLEY: These cases are of interest because of the presence of massive cerebral hemorrhage in two young people; both had unbearable headaches and increasing intracranial pressure with rapidly developing choked disks, which required operative interference. Neither case showed evidence of a neoplastic or inflammatory cause. I am inclined to the opinion, however, that an encephalitic virus was a prominent factor in the etiology, this deduction being reached from my experience with three epidemics of encephalitis that followed closely after epidemics of influenza; and, further, evidence of hemorrhagic disease of the new-born appearing in the Worcester Hospitals coincident with the encephalitis. Further data of a predisposition to hemorrhage following influenza and encephalitis will be awaited with interest.

DR. E. W. TAYLOR: As Dr. Burley says, he was good enough to ask me to see his second case in consultation. The evidence of tumor seemed to me, and I think also to him, to be slight in consideration of the very rapid onset of the symptoms and the general appearance of the patient, in spite of the fact that she had a rapidly increasing choked disk with ultimate complete paralysis of the third nerve on one side. The possibility of a latent glioma with sudden hemorrhage leading to increased pressure was considered. One of the striking conditions on the clinical side was the blood-stained spinal fluid after most of the punctures, with a tendency to become deeper as the fluid was withdrawn. The massive ventricular hemorrhage must have occurred very shortly before death, in spite of the fact that the blood in the fluid would seem to point to the escape of blood into the spinal canal earlier. The microscopic examination of the area in the temporal lobe showed no tumor. There is a certain amount of reaction around the hemorrhage, which would occur in any case of such an extensive lesion.

The conclusion which Dr. Burley has reached from the clinical observation of the case is that we have to do with an encephalitis and, as he also suggests, hardly of the epidemic form. Certainly on the clinical side it is a very unusual development of encephalitis lethargica. It is possible that, when further sections are made, we may find evidence as indicated by Dr. Cobb's preliminary observations, of perivascular infiltration, which will bring it into the category of encephalitis. In the literature very small mention is made of hemorrhage as at all a constant pathologic finding in encephalitis lethargica.

Just a word about the first case, which appears to me still more mysterious. I happened to see this man also, when he was on the high road to recovery. It is difficult to see why he recovered through a simple decompression operation with the relief of his symptoms of intracranial pressure and without residuals of any sort. The cases taken together form a small group of hemorrhagic disease of unusual rarity, and it seems to me of very great interest.

DR. GILBERT HORRAX: The two cases are certainly of extraordinary interest, as Dr. Taylor has said. I was struck in the report of the second case with the fact that this woman had a complete ptosis and pupillary changes of the third nerve, and it occurred to me that we might be dealing with an aneurysm of the base that had suddenly burst. We have had several cases at the Brigham Hospital with similar neurologic signs. However, an examination was made at the base of the brain in this woman and no aneurysm demonstrated. In the first case it is a little problematic as to just what the boy had: whether the clots that came through the needle were due to trauma to the cerebral vessels or from a large clot. The lack of brain pulsation with marked tension

at the time of operation would indicate the latter. The fact of the blood filling up the ventricular system early does not seem to me at all inconsistent. The natural assumption is that such filling up would lead to sudden death, but sometimes it may go on for a long time, through the ventricles and down the cord, before causing death.

DR. STANLEY COBB: The specimens I looked over this morning indicated an encephalitis, not of the lethargic type.

DR. H. R. VIETS: I should like to ask if these cases are not somewhat analogous to the cases of spinal cord hemorrhage coming on acutely which Dr. Burley reported several years ago: acute ascending myelitis.

DR. B. T. BURLEY: Replying to the question of Dr. Horrax as to the need of operative interference in Case 1, I would call attention to the presence of rapidly increasing choked disk in the right eye, the left having been previously lost, as sufficient reason for an exploratory craniectomy with subtemporal decompression.

Regarding the question raised by Dr. Taylor as to rapid fatality from ventricular hemorrhage, these cases, particularly Case 2, showed the presence of fresh blood from lumbar puncture consequent on fresh cerebral hemorrhage, giving us evidence that hemorrhage into the ventricular spaces is not always promptly fatal.

Replying to Dr. Viets, there is an interesting analogy in the different types of acute ascending myelitis and the different types of encephalitis, particularly those with and without hemorrhage.

AN UNUSUAL TUMOR OF THE THIRD VENTRICLE IN A CASE OF DIABETES INSIPIDUS. PERCIVAL BAILEY and B. J. REIFENSTEIN.

Anatomic specimens from a case of diabetes insipidus were presented, the history of the patient being as follows:

G. H. C., a schoolboy, aged 16, with essentially negative family and past history, was admitted to the medical service of the Peter Bent Brigham Hospital, Nov. 9, 1921, complaining of thirst and frequency of urination. The patient stated that three months previous to entry he had a sudden onset of thirst, and had since drunk about five quarts of water daily, with a corresponding increase in the amount of urine passed. He was obliged to get up three times or more each night to urinate. In addition, he was very constipated and had lost about five pounds in weight.

The boy was poorly nourished, with a diffuse brownish pigmentation all over his body. The tendon reflexes were very active, with a tendency to clonus at each ankle. The Wassermann reaction was positive in the blood; the spinal fluid was normal. Antisyphilitic treatment was instituted and continued vigorously, and the polyuria was controlled fairly well by intranasal insufflation of pituitary extract.

July 17, 1922, the patient was again admitted to the medical service complaining of persistent frontal headache which had been present for two months. For two weeks previously he had had weakness of the legs, and transitory diplopia. For the four last mornings he had vomited his breakfast. He was at this time drowsy. There was an extensive acneiform rash over the body; the reflexes were very brisk; and there was general constriction of the visual fields. He was transferred to the surgical service July 21, 1922. At that time he weighed 87 pounds. His fluid intake was 1,900 c.c. and output 1,500; the

metabolic rate was —38. The blood Wassermann reaction was still feebly positive. He was very emaciated and drowsy. Roentgen-ray examination showed the sella turcica to be normal.

The patient became rapidly more feeble and on July 29, it was noted that the pupils were absolutely fixed to light and in accommodation. The left pupil was widely dilated and there was ptosis of the left eyelid. The left plantar reflex gave a dorsal response. The next day, the right pupil was also dilated and there was a slight ptosis of the right eyelid. From that time he became very weak. No further neurologic symptoms developed. On August 19, he weighed 72 pounds and his urinary output had fallen to 400 c.c. August 21, his rectal temperature had fallen to 95 F. and August 24, he died, with a terminal hyperthermia.

At necropsy, the pineal gland was found to be about twice the normal size, and in the third ventricle was a very vascular tumor, spherical, about 4 cm. in diameter. The pituitary gland was normal. The other organs of the body, especially the kidneys, were normal.

We wish to call attention to the fact that the case was not operated on because the usual symptoms of a suprasellar tumor were absent, as were also general pressure symptoms. The location of the lesion was certain because of the diabetes insipidus. Clinically, the course of the disease simulated closely a basilar syphilitic meningitis. The histologic structure of the tumor was very unusual, but a positive opinion as to its nature and origin will have to await further investigation.

DISCUSSION

DR. E. W. TAYLOR: I should like to ask what is considered to be the cause of the cessation of the diabetes insipidus—the spontaneous disappearance of that symptom.

DR. PERCIVAL BAILEY: That is a very interesting phenomenon, which I cannot explain. It is frequently seen in these patients before death. It is not likely to be solved until we know much more concerning the essential mechanism of diabetes insipidus.

DR. E. W. TAYLOR: Is it possible that the growth of the tumor caused the cessation of the diabetes insipidus, since, as the tumor increased, the diabetes diminished? Was it a sufficiently destructive lesion to lead to the cessation of the extreme urinary output?

DR. PERCIVAL BAILEY: It is difficult to answer these questions because of the inherent difficulty of determining whether any neurologic symptom is due to an irritation or to the removal of an inhibition. In this case the tumor had invaded the base of the brain along the perivascular spaces of Virchow-Robin and had also invaded the meninges around the cerebellum and the pineal gland. It may be that this increasing invasion led to a cessation of the polyuria. I have noticed in the case of dogs who have had a very destructive lesion of the hypothalamus that as long as they are lethargic the polyuria is absent and only appears as they gradually recover from the lethargic condition; if the lesion is less destructive the polyuria appears immediately.

FACTS AND FANCIES OF MENTAL HYGIENE. DR. GEORGE V. PRATT
by invitation.

Misinformation and confusion as to what the mental hygiene organizations are endeavoring to do sometimes appears in medical groups as well as among the laity. As perhaps the latest of the nation's great public health movements,

mental hygiene is just commencing to find its feet. It is neither a complete nor an exact science like bridge building or analytic chemistry. Its principles are not as yet sufficiently developed to permit of predicting accurately the degree and kind of response to stresses, strains, or emotional situations. It is not a kind of sect or fad or a new cult. It does not advocate a new faith or system of treatment similar to osteopathy or the electronic reaction of Abrams. It has no special technic or definite system of therapy and is not held out as a panacea for all the mental ills of the community. Instead, mental hygiene feels that, like psychiatry, it possesses a little knowledge concerning the nature, cause, and more especially, the prevention of some, not all, mental disorders. It believes this knowledge, admittedly incomplete, is nevertheless sufficient to warrant its withdrawal from laboratory seclusion and its application to actual community problems.

Mental hygiene, if it ever was, is not now in the closely guarded control of a few persons. Today mental hygiene embraces not only the science of medicine, but its practical application brings it into close touch with almost every phase of human endeavor. Thus it contacts fields of sociology, economics, criminology, and so on. Mental hygiene as a public health movement does not endorse unreservedly, extreme viewpoints on either psychoanalysis or psychology. It finds value in the products of both of these schools of thought, but only as adjuncts to a program of treatment, and not the entire program itself. State Mental Hygiene Societies have widely different programs. Some engage in clinical work almost entirely, while others devote their activities to educational work. In Massachusetts where an elaborate state hospital system of out-patient clinics exists, the Mental Hygiene Society engages exclusively in educational work.

The Massachusetts Society for Mental Hygiene functions on the belief that if early detection of early symptoms of approaching mental disorder is a desideratum for prevention, then there is implied on the part of the public a knowledge of what these symptoms are. But until some of the fog of stigma, superstition, and ignorance that surrounds the mentally sick is dispelled, little can be hoped for in the way of patronage of out-patient clinics. Methods of education are numerous: In the Massachusetts Society for Mental Hygiene they include a Lecture Bureau Service supplying speakers; frequent public conferences; publication of simply worded literature; issuing of a Monthly Bulletin; insertion of courses in mental health training into the curriculums of normal schools, nurses' training schools, schools for social work, and the like. Recently, attention has been concentrated on conducting instruction or lecture courses of six to eight weekly lectures for such specialized groups as school teachers, social workers, and nurses. Much attention has also been given recently to focusing interest on the problems of childhood, in the formative, flexible age when good mental habits may best be instilled and faulty ones corrected.

DISCUSSION

DR. BRONSON CROTHERS: In writing a paper on mental hygiene, I wrote as an admitted amateur and outsider who had made an earnest attempt to understand the campaign. The question that came up for chief discussion was the wisdom of attempting to popularize the freudian and the more extreme psychologic points of view. In particular, I regretted the publication of books for mothers based on freudian theories and asked for authority for the statement that 50 per cent. of mental disease could be prevented by proper recognition and early treatment of conflicts. That statement, made in the heat of

a campaign for recognition and financial support, is less aggressively repeated in literature sent out by the Massachusetts Committee which quotes the Governor's message to the effect that 50 per cent. of mental disease is preventable. Except for these criticisms of detail I am, of course, heartily in favor of mental hygiene. The reason for writing at all was to call attention to the movement as a whole and to emphasize my own feeling that mental hygiene is not, as many feel, simply a division of psychiatric work. Obviously complete and whole-hearted support of a program which can be agreed on is essential. In order to stir up discussion on this subject it seemed worth while to present the reactions of an outsider to the fascinating and important series of articles published by the National Committee.

DR. E. STANLEY ABBOT: It seems to me that mental hygiene is a part of general hygiene. Just as we try to build up the bodies of children and make them as healthy as we can, so the object of the mental hygiene movement is to understand what is necessary for the best development of the mental activities of the child. The mental hygiene movement is trying to find out what the principles are that lie back of all behavior as guided by our mental activities, and it is for that reason perhaps, because the Freudians have done more to analyze the mental factors of behavior, that we turn rather to the freudian psychology for help. Certainly academic psychology has done very little to help in this field. Mental hygiene has seen the need of studying these mental factors in behavior, and it so happens that the Freudians have done more than any other group to try to analyze the basis of behavior and of our mental reactions in relation to them. That does not mean, however, that mental hygienists have to swallow freudian psychology bob, hook, and sinker. I do not think any of the men in the movement really do so. It would be unwise to do it, but they do reach out in that direction to get what help they can because help does not seem to be coming from other directions.

DR. BRONSON CROTHERS: The question is whether freudianism can be propagated by elementary discussion among them.

DR. E. STANLEY ABBOT: I do not think it can. I deprecate very much the entrance into alleged freudianism of half-baked laymen, clergymen, psychologists perhaps—various helpers of humanity who have no solid medical or psychologic background. I do not feel they belong to the mental hygiene movement. They see an economic opportunity in writing on those subjects. It is said that Freud's introduction is one of the best sellers, and many who are not qualified write books to take advantage of its vogue.

DR. PERCIVAL BAILEY: It is a question of fundamental importance to determine just how much of the theory and practice of medicine it is wise to teach the public. Every physician should give the matter earnest thought, especially at the present moment when the new journal, *HYGEIA*, is being launched. The future policy of this magazine as well as of the journal of the Society for Mental Hygiene, will depend largely on the reaction of the medical men to the material published in it. The statement that we could prevent 50 per cent. of functional nervous disease in children if we could avoid the mental conflicts which gave rise to them does not shock me. I think the figure is probably too low. I am sure, also, that Janet, who cannot be accused of being a Freudian, would take no offense. The problem of determining the nature of these conflicts and the means of avoiding them is still far from solution, although great progress has been made.

DR. PRATT: Psychiatrists giving popular mental hygiene talks to lay groups are frequently asked afterwards, "What can you do for a child that does so and so?" It is sometimes difficult and not a little embarrassing to explain that it is impossible to offer blanket suggestions for the correction of specific behavior or conduct disorders in children. Unlike the case of the mother who brings her child to the physician for some well recognized physical defect and for which a treatment program can be outlined that would be equally applicable to a dozen cases presenting the same symptoms, one must first in this work discover the particular etiologic factor that produces the trouble. I mean, for example, that three children all presenting the general symptoms of "tantrums" and lack of discipline will almost certainly all have distinctly individual and widely differing reasons therefor. This is why we stress in public lectures that the treatment of mental or nervous conditions is a highly individual process and that each case is a law unto itself.

Another thing that embarrasses the progress of mental hygiene work in the community is the tremendously deep feeling of stigma that attaches to the whole subject of mental disease in the minds of a majority of the public. The dispelling of this stigma and fear is perhaps one of the most difficult immediate things confronting the mental hygiene organization. It prevents the early incipient case from receiving the prompt treatment that might abort permanent invalidism and it causes many people to hide their mentally sick as if mental disease were a disgrace. A woman wrote to my office this morning asking for information about the care at home of her mother who is destructive, badly hallucinated, and with a number of uncomfortable delusions. After admitting that the family physician advised institutional care this woman went on to say that, "Of course you understand mother is not crazy, but is only a little nervous and we would not dream of putting her away in an asylum." The alleviation and ultimately the dispelling of some of this fog of fear, superstition, and prejudice is a most important objective in the work of the Massachusetts Society for Mental Hygiene.

It is important to realize that various state mental hygiene societies have widely differing programs. Thus in Connecticut, the State Mental Hygiene Society finds its chief activity in the field of clinical work. On the other hand here in Massachusetts where there exists a widespread and splendid system of state hospital out-patient clinics, the Massachusetts Society for Mental Hygiene has sharply limited its work to the field of education and publicity. The work of the State Division of Mental Hygiene in establishing and conducting clinics, and the work of the Massachusetts Society for Mental Hygiene in performing important developmental work in a community along the lines of education, dovetail together nicely.

One thing I wish to bring out in conclusion: We have at times been mildly criticised for making the general statement that "Mental Disease is preventable; mental health is procurable." We admit that we have made this statement more or less of a public slogan and we also admit that, technically speaking, this statement is not 100 per cent. accurate. The precise amount of mental disease that can be prevented we do not know. Theoretically we all know that general paralysis is preventable through absence of syphilitic infection. I think we are also ready to admit that, so far as we know at present, dementia praecox is not preventable.

We feel, however, that the amount of positive knowledge in this matter, admittedly incomplete, is nevertheless sufficient to warrant making a start

toward public education in the maintenance of good mental health. The Mental Hygiene Society has been and continues to be interested in not offering mental hygiene as a panacea for all the nervous and mental ills of the community. We publicly stress the limitations of knowledge on this subject and are extremely anxious to avoid being linked up in the public mind with the "applied" psychologist, the commercial psychoanalyst, the "new thought" exponents, and other tassels on the lunatic fringe.

Perhaps the most valuable single factor contributing to the advance of community mental hygiene has been the establishment of the habit clinics which Dr. Douglas A. Thom first instituted at the Baby Hygiene Station a year ago. Such clinics, dealing with preschool age children who are presenting early symptoms of personality defects or behavior disorders, are extremely valuable in the maintenance of good mental health, and, of course, the avoidance of later mental disorder as well.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, March 6, 1923

EDWIN G. ZABRISKIE, M.D., *President*

THE PRACTICAL BEARING ON DIAGNOSIS, OF THE PHYLOGENY AND CLASSIFICATION OF SPINAL MOTOR INTEGRATIONS. DR. WALTER M. KRAUS.

The factors necessary to produce postures of the trunk and extremities in normal man are at least three: (1) Determination of the positions of the various parts of the trunk and extremities by means of reciprocally controlled antagonistic muscles. The endogenous spinal fibers by linking peripheral motor neurones, play the major part in this determination of postures. Examples of postures thus determined are the position in opisthotonos and of the leg in paraplegia in flexion. (2) Activation of the neurones which determine posture. This activation is brought about by afferent impulses entering the spinal cord through the posterior roots and by impulses of supraspinal origin, probably arising in large part in the motor basal ganglions, but also in masses of grey matter situated more caudad. When these impulses are permitted to be continuous and increased beyond normal, due to loss of control by superior controlling systems, spastic or hypertonic states result. When these impulses are discontinuous, rhythmic or arrhythmic movements result. (3) Maintenance of posture by the synergic control of the cerebellar labyrinthine mechanism.

Determination, activation and maintenance are separate physiologic activities and originate in separate anatomic mechanisms of the nervous system. The endogenous fibers of the motor portion of the spinal cord determine all of the postures of the trunk and extremities, except that of erect posture and that which occurs in the arm in cerebral hemiplegia. These two latter postures are most probably of supraspinal origin.

When muscles are activated by means of the action of the corticospinal tract (upper motor neurones of the pyramidal tract) on anterior horn cells, the postures determined at physiologically lower levels are destroyed.

In order to determine the endogenous spinal connections which correlate the various segments of the anterior horn cells of the spinal cord, it becomes necessary first to consider the anatomic arrangement of these cells in man as well as in lower animals. It has long been recognized that they are divisible into two large groups—one mesial and having to do with the axis, the other lateral and having to do with the extremities. The mesial group, furthermore, is divided into two subgroups, one dorsal, the other ventral, which supply the corresponding dorsal and ventral axial muscles (Tilney and Riley). The lateral parts of the anterior horn cell columns are present in the cervical and lumbosacral enlargements and have to do with the extremities. According to the best authorities, the localization of these cells is such that the most distal (ventral) muscles are controlled by the most dorsally placed cells.

The movements of the trunk are brought about by synergic contraction of the dorsal and ventral muscle masses on the two sides in various combinations. However, first, the anterior horn cells controlling either ventral or dorsal muscles on one side are linked by intersegmental and intrasegmental fibers. Further linking of these groups, one to another, dorsal with dorsal, ventral with ventral, or dorsal with ventral on either right or left sides, is also present. When the dorsal and ventral group on one side are made to contract in unison and in excess of the corresponding group on the other side, lateral movements are produced. On the other hand, when the right and left dorsal muscles are made to contract in unison in excess of the right and left ventral muscles opisthotonic reactions result; while, per contra, when the right and left ventral masses are made to contract in unison in excess of the right and left dorsal masses, emprosthotonic reactions result. This leads to the possibility of classifying some of the integrating functions of the endogenous fibers of the spinal cord as follows:

Unilateral axial	Bilateral axial
(a) Dorsal (right and left)	(a) Dorsal
(b) Ventral (right and left)	(b) Ventral
(c) Dorsoventral (right and left)	(c) Dorsoventral
(d)	(opisthotonos, emprosthotonos)
	(d) Dextrosinistral

An analysis of the integrative control of the extremities can best be made on the basis of the various phases of normal reflex stepping (Archives of Neurology and Psychiatry 9:184, 1923). There are two pairs of corresponding opposite sets of activities: the simpler pair representing movements of animals with singly hinged appendages (fish) and represented in man by pendulum-like movements at the shoulder and hip; the second pair representing the phases appearing in terrestrial animals. These two latter phases have already been described by Sherrington and others as the flexor and extensor phases of reflex stepping. They are superimposed on the first pair and conceal it except at two stages. The muscles of the extremities are familiarly known to be divided into dorsal and ventral groups. In the first two phases of reflex stepping showing movements at the shoulders and hips, both the ventral and dorsal masses acting at these joints are each separately integrated by the endogenous fibers of the spinal cord. The anterior horn cells of the ventral group are made to act together as are those of the dorsal group. Further integrative activity combines the dorsal and ventral antagonistic muscles by reciprocal innervation to make coordinated movements possible. Furthermore, the limbs on the same side of the body, as well as the pairs of fore and hind

limbs, are similarly linked. This makes the following classification of integrating functions possible:

Primary

Unilateral Appendicular	Bilateral Appendicular
(a) Dorsal (right and left; one limb)	(a) Dextrosinistral (fore limbs)
(b) Ventral (right and left; one limb)	(b) Dextrosinistral (hind limbs)
(c) Dorsoventral (one limb)	(c) Dextrosinistral (all limbs)
(d) Dorsoventral (fore and hind limb; right or left)	(d) Dorsoventral (all limbs)

The third and fourth phases of reflex stepping represent an alternation, at the three great joints, of activity of dorsal and ventral muscles. During walking, the act of carrying the leg from the backward to the forward position by means of flexion, represents the third phase, while the act of support represents the fourth phase. The positions of paraplegia in flexion and paraplegia in extension also illustrate these two phases. The integrating activities of the spinal cord determining these positions may be designated as follows:

Secondary

Unilateral Appendicular	Bilateral Appendicular
(a) VDV (right and left; one limb)	(a) Dextrosinistral (fore limbs)
(b) DVD (right and left; one limb)	(b) Dextrosinistral (hind limbs)
(c) VDV—DVD (right and left; one limb)	(c) Dextrosinistral (all limbs; normal reflex stepping)
(d) VDV—DVD (fore and hind limb; right or left)	(d) VDV—DVD' (all limbs; decerebrate and decapitate postures)

When all four extremities are set into posture by the ventral muscles acting at the hip and shoulder, by the dorsal muscles acting at the elbow and knee, and by the ventral muscles acting at the wrist and ankle and below, the animal is in the extension position of decerebrate posture; while, when the antagonistic muscles are active, it is in the flexion position of decerebrate posture. That both of these corresponding and antagonistic positions are characteristic of decerebration has been recently pointed out by Bazett and Penfield. The evidence that these postures are spinal in origin and dependent on the activity of the endogenous fibers of the spinal cord is easily found by examination of the work of Sherrington, Von Monakow, Riddoch, and l'Hermite in cases of complete division of the spinal cord in animals and man. Further proofs have already been given elsewhere by the speaker.

A lantern slide demonstration of the positions of the trunk and extremities in various nervous diseases illustrated the above described integrating activity of the endogenous fibers of the spinal cord in a number of different diseases of the nervous system.

DISCUSSION

DR. SMITH ELY JELLIFFE: Dr. Kraus has set before us a scheme of the analysis of voluntary motor function which is becoming more insisted on. Considerations concerning walking and postures have been discussed largely as matters of general description and such discussions have lacked precise, dynamic concepts. Dr. Kraus' presentation is highly desirable in that he has shown that we must be more specific in our descriptions and state where the impulse arises and where it goes.

There are one or two points to which I would like to call attention and which have some connection with the work of Tilney and Riley as set forth in their book, which Dr. Kraus has quoted. They speak of an impulse arising within a motor cell and going to another motor cell. No impulse arises in a motor cell. Every anterior horn cell is only a part of a reflex arc. The impulse

comes from the outside, and the idea of its autonomous origin in the cell is a faulty concept. The spinal cord never exists by itself and cannot be explained on the basis of its own activities. It must be related to external environmental factors.

I do not think that the concept presented by Dr. Kraus regarding the columns is correct. We cannot insist too dogmatically on the continuity of columns, which, in reality, are more or less broken or segmental. The cell groups are arranged in columnar form, but one cannot speak of the function of a column for a column has no function, although different segments within the column have partial functions in the handling of reflex activities. My interest in the localization of muscle groups has extended to attempts at explaining the breakdown in various types of integrations in cases that are not fully understood. It may be remembered that I have expressed views on the connection between vascular integrating factors and muscle group integrating factors in multiple sclerosis of certain types, and have tried to show that unconscious wishes on the part of the individual to bring into effect certain types of activity on the environment (kicking the world in the face, for instance) gives rise to changes in the muscular and vascular integrative factors, which may determine a breakdown in parts of the machinery, related to those that Dr. Kraus has brought forward.

In connection with the subject of exposure, I cite an observation: A patient was brought to my office with numerous records setting forth that she was subject to an hysterical limp. I noticed certain affective reactions as the patient walked and I asked whether a roentgenogram had been taken and was told that it had not. When it was done it showed what appeared to be a myeloma, but this was later reported to be a giant cell sarcoma, of a non-malignant type, at the lower end of the left tibia. I took considerable interest in the genesis of the giant cell sarcoma and asked myself how this could have occurred, as I believe that all problems in medicine have a neurologic basis. I first told her husband that the difficulty was surgical.

In the process of investigating her general psychic state, it soon became apparent that she constantly held the left leg, night and day, in a continuous postural attitude. Study of the unconscious showed that this posture served two purposes: first, partially closing the vagina from attack from the external world, that is, unconsciously protecting herself from sexual assault; and second, keeping the labia together in order to produce masturbatory activity. It would be interesting to find out what muscles are involved in such a postural attitude and what relationship these muscles have with the ligaments at the lower end of the tibia concerned in the pull of the chronically used muscles. Further, what is the relationship between the chronic stimulus of the ligamentous play on the bony structures which we know are inter-related? Is it possible that a chronic postural attitude can have relation to a chronic inter-related reaction of the ligaments, producing an irritation in the underlying bone and causing a giant cell sarcoma?

DR. W. M. KRAUS (closing): It is, of course, true, as Dr. Jelliffe has remarked, that impulses do not arise within motor cells, but are dependent on impulses afferent to the motor cells. I have not discussed the relation of the extent of these columns to embryologic groups of muscles, since that is an extensive subject. The existence of a column of cells depends on the appearance in the course of evolution of a new group of muscles. The mere fact that several lateral columns of cells have been added to the mesial columns, where limb control has become necessary, indicates this. A definite column of cells

has only the functions of controlling, subservient to afferent impulses, the particular muscle it serves, and so far may be said to have a definite function.

As to the genesis of a giant cell sarcoma on the basis of postural reflex, that transcends my powers of imagination. But the muscles and ligaments involved in the production of any given posture may be described without difficulty.

REMARKS ON THE HISTOPATHOLOGY OF AMAUROTIC FAMILY IDIOCY. DR. JOSEPH H. GLOBUS.

In the course of an anatomic study of the infantile (Tay-Sachs) and juvenile (Vogt-Spielmeyer) forms of amaurotic family idiocy, the following observations were made: (1) The total or partial cell changes, as described by Shaffer, form the background for all forms of this disease. (2) These common features, as well as the occurrence of transitional forms, do not allow of the separation of the disease into anatomic groups, but permit only a division into clinical forms. (3) Contrary to the general impression and repeated statements that the disease process is limited to the gray matter and involves every ganglion cell of the cerebrospinal axis, it can be said that the white matter in many instances is equally involved and that many cells in the gray matter may escape the disease process. (4) The frequent involvement of the cerebellum must be looked on as extension of the same pathologic process and not as another added feature to the disease. (5) Each case, or group of cases, may have its peculiar staining reactions. This staining variation is simply an expression of the chemical character of each case or group of cases. (6) In the complete absence of inflammatory changes, and in the presence of all the earmarks of a degenerative lesion, it may be concluded that the disease is degenerative in character and due to some metabolic disturbance.

The article was illustrated with lantern slide demonstrations.

DISCUSSION

DR. I. STRAUSS: While there are variations in the disease process, the fundamental changes are present in all cases of this kind. Those of a secondary nature may vary in intensity and distribution. In the case which I studied, and to which Dr. Globus has referred, I observed very little change in the white matter. The spinal cord showed involvement of the anterior horn cells, but no change in the fibers of the cord when stained by the Weigert-Pal method. The interest to me in this disease rests in two factors. In the first place, this variety of disease (including the forms described by Vogt, Spielmeyer and Sachs) is one of the few diseases of the central nervous system that has a characteristic histologic picture, which makes the diagnosis of the disease absolutely positive. Another point about the disease is that we are dealing with something that is very fundamental in the organism, namely, with changes in the germ plasm, as well as factors introduced of an endogenous character. It has not been discovered what it is that causes such tremendous destruction of the cellular elements. In all the cases one notices that the children are remarkably well developed infants and show no signs of paralysis. Even if they do not die because of the disease, but from intercurrent infection, one finds a very extensive disease process and one wonders how the function of these cells can persist to the high degree in which it does, in spite of the remarkable morphologic change and destruction. It points out one fact, and that is, that when there occurs what appears, morphologically, to be a very

marked alteration of the cell, still there is enough function apparently going on in that cell to maintain the processes of life. Therefore there is something in the functional activity of the cells of the central nervous system of which we know very little at the present time; and they cannot be measured by their morphology. I am associated with another worker in the study of these brains from a chemical standpoint and it may be that we shall find definite chemical changes in the constituents of the brain. That, of course, will only be another side-light on the question. I think the actual discovery of the cause of this remarkable disease is something which is going to baffle us for many years, until we know something more regarding the actual nature of the function of the central nervous system cells.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 3, 1923

E. G. ZABRISKIE, M.D., *President, in the Chair*

MEMORIAL TO WILLIAM M. LESZYNSKY, A.B., M.D. DR. J. RALPH JACOBY.

We greatly regret the recent death of Dr. William M. Leszynsky who was for many years intimately associated with the work of this Society, was one of its former presidents, and was always an earnest and untiring co-operator in its various activities. Dr. Leszynsky was born at Newport, Rhode Island, June 16, 1859. After leaving school he took the scientific course at the College of the City of New York, where he received the degree of Bachelor of Arts. In 1876, he entered the University of New York Medical School, and received his degree of Doctor of Medicine in 1879. At the time of his death, March 3, 1923, he was Attending Neurologist to the Lebanon Hospital and the Manhattan State Hospital, Consulting Neurologist to Christ Hospital (Jersey City), Harlem Hospital and the Peoples' Hospital. He was for many years Consulting Neurologist to the Manhattan Eye and Ear Hospital and the Demilt Dispensary, and Lecturer on Mental and Nervous Diseases at the New York Post Graduate Medical School.

Dr. Leszynsky was a member of the New York Neurological Society, the New York Academy of Medicine, American Medical Association, American Neurological Association, Medical Society of the Greater City of New York, Society of Medical Jurisprudence, Metropolitan Medical Society, New York Physicians' Association, New York Medical Union, Harlem Medical Association, Manhattan Medical Society, Medical Society of the County of New York, New York Medico-Surgical Society, Physicians' Home, Society of the Alumni of Lebanon Hospital, Association for Research, National Committee for Mental Hygiene, Thomas Hunter Association of Grammar School No. 35, etc.

Dr. Leszynsky was a prolific writer and contributed many scientific articles to medical literature. His attainments in the field of neurology were broadly recognized. Here, in New York City, his endeavors were most appreciated, because we were able, as his associates, to come more closely in contact with his personality and to know the human side of him. In our Society we came to know him not only as a man of precise knowledge and high scientific

attainments, but also as one so modest and retiring that the very qualities that made him preeminent in his profession sank into the background of his geniality and personal charm.

The New York Neurological Society has passed a resolution that a copy of this Memorial be sent to his widow, with expressions of deepest sympathy; and that copies also be forwarded to the Journal of Nervous and Mental Disease, the Archives of Neurology and Psychiatry, and the New York Medical Journal and Medical Record.

LUMBAR ARTHRITIS AS A CAUSE OF SCIATICA. DR. P. WILLIAM NATHAN (by invitation).

The symptoms of sciatica, as usually described, cannot be correlated with an actual lesion of the nerve trunk or the sacral plexus. The neurologic symptoms are evidently radicular and are more or less exactly similar to those that are known to accompany lesions of the vertebrae and the cauda equina. The spasm of the muscles, the deviation of the spine and the fact that the symptoms are aggravated by movement and alleviated by rest, strongly suggest that the lesion is of an inflammatory nature involving the lumbar vertebrae.

In an endeavor to confirm this a priori conclusion, 140 consecutive cases of so-called sciatica were examined roentgenographically at the Mount Sinai Hospital and in private practice. Of these, 126 showed more or less extensive arthritic changes in the lumbar vertebrae. It may, therefore, be concluded that in a large majority of cases with sciatic syndrome, the cause is to be sought in inflammatory vertebral lesions. It is, however, necessary and at times difficult, to exclude tuberculous and malignant diseases in this region.

DISCUSSION

DR. C. C. BELING: In what position were the roentgenograms taken, upright, or lying down?

DR. NATHAN: They were taken lying down.

DR. BELING: Dr. Nathan has presented a group of cases of sciatica due to vertebral disease, but some cases are due to muscular changes. In 1904, Gowers stated that some cases of sciatica were due to inflammatory conditions of the white fibrous tissue of the muscles, and thought that the spindle cells of the muscles and tendons were affected. When we speak of pain on the outer aspect of the thighs it is due to lack of differentiation of the location of the pain. Sometimes affections of the external lateral cutaneous nerve are mistaken for sciatica. Sciatica is simply a symptom-complex that may be due to many conditions, and one of the commonest causes is doubtless neuro-muscular fibrositis.

DR. ROSENBERG (by invitation): I have seen many cases at the hospitals and also in my office. One thing, which I have always insisted on, is the roentgenographic examination of the hip and spine. Although many cases have been studied, the report of the roentgenologist has always been negative; in fact, I can hardly recall a case in which the vertebrae have been shown to be involved. Occasionally sacro-iliac dislocation is reported, but not one case where the vertebrae are involved. The pictures shown by Dr. Nathan indicate definite vertebral involvement. Clinically, one sees cases of so-called sciatica with definite scoliosis, which, with comparatively short or light treatment, are relieved of pain and at the same time of the scoliosis. That has

been observed many times. The patients seem to suffer from a muscular contracture that causes the scoliosis. That is why I also need to know in what position the roentgenograms were taken. I have seen scoliosis with an appreciable difference in the measurements of the hip. The orthopedic condition disappeared after an epidural injection. One is puzzled to know how the condition, which apparently is one of vertebral involvement, can be affected by an epidural injection.

DR. NATHAN: I cannot say much in regard to the first speaker's remarks. I have never been able, in the examination of a large number of patients, to demonstrate a condition which resembles so-called fibrositis. Patients often complain of pain at the origin of the muscles, and myositis is not unusual, but one is not able to demonstrate that it acts in such a universal manner as has been claimed. Dr. Rosenberg's view-point regarding recovery from sciatica is well taken. There are a considerable number of cases that come in as sciatica, having scoliosis, etc., which clear up after epidural injection. This is perfectly true. Lumbar spondylo-arthritis, like other joint conditions, may be mild, or it may come on acutely and disappear with or without treatment. In some cases the scoliosis is due simply to muscle spasm which disappears with the subsidence of the inflammation; in others it is due to bony involvement and destruction of the tissues; but even these, when treated early, are amenable to orthopedic treatment. With a well-equipped roentgen-ray laboratory, and the services of a good roentgenologist, changes may be detected in the vertebrae in many cases, although in some they are not marked. In only fourteen of 140 cases were we unable to detect any vertebral change. The point I wish to bring out is that the syndrome, sciatica, cannot be brought into correlation with a condition in the sciatic nerve trunk. The symptoms as they are described and as they are encountered in practice, are characteristic of root irritation and, as can be demonstrated, this root irritation is most often induced by lumbar or more extensive spondylo-arthritis due to nontuberculous infection. However, as malignant and other diseases in this region induce similar symptoms, these must be carefully excluded.

BARBITAL INTOXICATION. DR. IRVING J. SANDS.

This report analyzes a series of fifteen cases illustrating the various manifestations of acute and chronic barbitol poisoning. The drug primarily causes dilatation of the small vessels and capillaries, producing a slowing of the circulation, reduction in oxidation, heat dissipation, and a lowering of the temperature. The slowing of the cerebral circulation and the reduction in the rate of oxygen and carbon dioxid exchange in the cerebral capillaries are responsible for the hypnotic effect of the drug. In small physiologic doses, about 90 per cent. of the drug is recoverable from the urine; in larger and in toxic doses only about 50 per cent. is found in the urine, the remainder being stored in the tissues of the body.

Acute barbitol intoxication results from the imbibition of a large dose of the drug, usually over 50 grains. The individual is comatose; the temperature is subnormal or moderately elevated; the pulse is slow or rapid; and the respiration may be lowered or increased. The face is flushed, the lips and finger tips are cyanosed, and the extremities are cold and clammy. The blood pressure is low; the urine contains albumin, granular and epithelial casts, and barbitol. The tongue is heavily coated and the breath foul. There may be incontinence of urine and feces. The superficial as well as the deep reflexes

are diminished, and in the more serious cases may be absent. The pupils may not react to light or in accommodation. Spontaneous contraction and dilatation of the pupils are regarded by some observers as pathognomonic of barbitol intoxication. On regaining consciousness, the patient shows a thick, drawling, indistinct speech, which resembles the speech of the general paralytic. There may be diplopia and nystagmus is common. Delirium may supervene. Death usually results from bronchopneumonia. At necropsy a general hyperemia of all organs is found, with cardiac dilatation, pulmonary edema, bronchopneumonia, degeneration of the convoluted tubules of the kidneys and fatty degeneration of the liver. Except for the presence of hyperemia, the brain is normal. Barbitol is recoverable from the various organs. Recovery of the individual from 125 grain doses has been frequently reported in the literature; larger doses usually prove fatal.

Chronic barbitol intoxication results from repeated small doses of the drug. The patient is somewhat cyanosed, and there may be erythematous skin lesions. The station and gait are ataxic and there is a general dyssynergia. Speech is drawling and indistinct; the breath is foul, the tongue coated and constipation is present. The body temperature may be subnormal, the blood pressure lowered and the pulse slow. Mentally there is an irritable, suspicious attitude and manner, emotional instability, defect in attention and poor retention. Memory, especially for recent events, is poor. Judgment is defective, and there is poor insight into the condition. These cases must be differentiated from general paralysis, epidemic encephalitis, uremia, and cerebral neoplasm.

The personality of individuals who suffer from barbitol poisoning is worthy of attention. Experience leads to the conclusion that the vast majority of persons with acute barbitol intoxication belong to the manic-depressive group of people, while those with chronic barbitol intoxication had the personality of constitutional psychic inferiority. Therefore one often finds that these patients take the drug with suicidal intent. Furthermore, the patients with chronic intoxication, take the drug as a flight from reality whenever faced with a situation that entails painful emotional feeling. Such people often take alcohol, and many of the chronic barbitol intoxication cases occur in alcoholics. Several of these patients admitted drug addiction as well.

The limitation of the sale of barbitol to physicians' prescriptions, and the use of the term diethylbarbituric acid, rather than the trade name veronal or barbitol would materially limit the number of poisoning cases.

DISCUSSION

DR. S. R. LEAHY: Dr. Sands has covered the ground very well. I disagree, however, in some minor details. I do not think the coma is characteristic of this condition; all drug intoxications are similar in that respect; also, it is not always possible to distinguish the coma from that of an organic disease. These people have often been users of others drugs: bromids, phenobarbital and others. I have seen one man with delusions of jealousy against his wife and great excitement, as the result of small doses. Every effort was made to prevent toxic symptoms and there may have been a psychosis mixed with the drug intoxication. I have seen a case with bromid intoxication, but the other features were not characteristic. In this case there was a history of depression ten years previously which lasted for several months. With the clearing up of the intoxication symptoms the woman developed a typical manic attack and had to be committed. These cases of barbitol intoxication are very

often diagnosed as general paralysis. The ataxic gait and reduction of the knee jerks suggest general paralysis. In every case it is necessary to study the serology. I am in hearty agreement with restricting the sale of this drug without prescription.

DR. W. J. DOUGHERTY: In regard to limiting the sale of barbitol as suggested by Dr. Leahy, the Board of Health recently added to the Sanitary Code a clause prohibiting the sale of barbitol without a physician's prescription; since then the number of cases of barbitol poisoning brought to Bellevue Hospital has been very small. Prior to the enactment of this restrictive clause cases of barbitol intoxication were always present on the wards of the alcoholic and psychopathic service of Bellevue; but now it is the exception to encounter cases of this type. One was received recently, the first in some weeks. This man formerly spent most of his time at Bellevue—literally going out by one door and in by another.

I do not believe that barbitol addiction is to be regarded as an expression of a manic-depressive psychosis, as one seldom finds manic-depressives who are barbitol addicts, although some depressed cases resort to the drug to terminate their existence. There is no doubt that barbitol addicts are, in most instances, examples of constitutional psychopathic inferiority, and that such persons feel incapable of facing the ordinary vicissitudes of life; they, hence, have recourse to barbitol in an effort to efface themselves from the scene for varying periods of time. They may be regarded as potential suicides, desiring to escape what seem to them the painful experiences of life, the suicide permanently, the addict usually only for the time being. Confirmation of this theory is to be found in the number of barbitol addicts who actually attempt suicide.

I have seen only one case of fatal barbitol poisoning. This man was found unconscious on the street with narcotics in his possession and was supposed to be suffering from morphin poisoning. His respirations were, however, too rapid for morphinism. The pulse was fairly rapid; the pupils were contracted, but not to the extreme degree observed in opium poisoning. He died half an hour after admission to the hospital; shortly before his death his respirations were but four a minute. This case did not exhibit the muscular twitchings that Dr. Sands has spoken of in fatal cases of barbitol poisoning. The case went to necropsy and the laboratory findings indicated that barbitol was the cause of death.

DR. B. ONUF: I have been much interested in regard to the relation of barbitol addiction to manic-depressive states. Dr. Sands finds acute barbitol poisoning mostly in manic-depressives; chronic poisoning, mostly in cases of emotional instability. Now, I do not know that we can make a sharp distinction between emotional instability and the manic-depressive psychoses. I think the manic-depressive temperament which is at the bottom of the latter, has many varieties. Some of its possessors are predominantly depressives, others prevalently manics, both often within physiologic limits. In still others there is an intimate mixture or alternation of these opposite trends and it is these who show particularly what can be termed emotional instability. In my writings on the manic-depressive temperament I have put down as one of the characteristics of this temperament or constitution, that those who possess it are very apt to become addicts to habit forming drugs, sometimes, as in the case of insomnia, for their narcotic and soothing effects; sometimes, as in the case of brain lag, for their exciting and efficiency increasing effects.

When the soothing and soporific effect is desired, barbitol is often preferred, for its milder action, to other narcotic or hypnotic drugs. I think this inclina-

tion to take habit producing drugs is illustrated by what we hear of conditions at Hollywood. This class of people can be described as being endowed mostly with manic-depressive temperaments; and drug addiction is said to be prevalent in that colony.

DR. E. G. ZABRISKIE: I am reminded of a general remark about conditions in China. When the authorities there made a strong campaign against opium, the consumption of gin increased 1,000 per cent. If we limit the selling of barbitol, what is the addict going to do next? We should consider these people as definitely of a psychopathic constitution, and attempt to guide them rather than merely restrict the sale of any particular drug.

DR. SANDS (closing): I have cases illustrating the points to which Dr. Leahy referred. Formerly, I invariably mistook these cases for general paralysis. I recall several patients whom I considered as subjects for the state hospitals, only to change my mind as soon as they disposed of the drug. In regard to Dr. Dougherty's statement, I came to the conclusion that these acute cases really did not want to die; what they really wanted was to get rid of their unhappy personality. In studying the entire "longitudinal section" of their life's reactions, it must be admitted that the acute barbitol intoxication cases are really manic-depressive individuals, while the chronic barbitol cases are examples of the emotionally unstable type of the constitutional psychopathic inferiority group of individuals. These patients have shown other peculiarities in conduct besides taking barbitol.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, March 23, 1923

CHARLES M. BYRNES, M.D., *President*

APOPLECTIFORM ONSET OF LEFT TEMPORAL LOBE SYMPTOMS PROBABLY DUE TO ENCEPHALITIS. DR. WILLIAMS B. CADWALADER.

The patient, 36 years old, is right handed, and had been entirely well until Jan. 2, 1923, when, while washing an automobile, he suddenly felt faint and weak. This was followed by a sharp pain in the left side of the forehead, after which he had to lie down and vomited repeatedly. He was immediately seen by a physician, who described his condition as one of confusion. In answering questions he repeated the word "lights," misplaced words and did not seem to comprehend. On examination the following day, he presented no physical abnormalities except that on being questioned he replied in a jargon-like manner, misplacing and misusing words, so that his speech was not understandable. (Jargon aphasia is the earliest manifestation of beginning word-deafness, and the improper use of words may precede distinct signs of word-deafness.) He could not carry out a simple command, such as to put one hand on his head or to open his mouth. He had no fever and no other evidence of intoxication or illness. There was no hemianopia.

Two days after the onset, ophthalmoscopic examination made by Dr. J. T. Carpenter showed paleness of the temporal portion of each disk. On January 19, the right optic disk was swollen 1 diopter, the left optic disk 2 diopters. At that time, I regarded this case as probably a left temporal lobe lesion, either

a tumor or encephalitis. He was transferred to the University Hospital, and the following positive signs were found: There was distinct word-blindness and he missed as many words in large type as in small type. Hearing for sounds was normal on each side, but there was distinct word-deafness. He could perform simple commands, but the execution of more complicated commands was impossible. The jargon aphasia had disappeared entirely.

January 25. Dr. de Schweinitz found: in the right optic disk 1.5 diopters of swelling; in the left optic disk 3.5 diopters of swelling with large hemorrhages on the nasal side, but no hemianopia. Blood count, spinal fluid, Wassermann reactions, and urine examinations were all negative.

January 30. The aphasia had improved and now resembled an amnesic type of asphasia. Word-deafness and word-blindness, though still present, were decidedly improved. Dr. de Schweinitz again reported the presence of bilateral choked disk, higher on the left. Spontaneous writing was impaired.

February 10. Dr. de Schweinitz reported 1.5 diopters of swelling in the right optic disk and 4 diopters of swelling in the left; this was practically the same as before, but he thought the appearance of the swelling was different from that of tumor.

February 15 and 21. Examinations by Drs. Halloway and Williams revealed that the swelling of the disk was diminishing.

March 1. Dr. Baer reported the swelling of the left disk as 1.5 diopters, and of the right disk as 1 diopter; on March 14 both disks showed about 1 diopter of swelling. Since then the swelling, as well as all other symptoms, has almost disappeared.

The case presented at first many difficulties in diagnosis. In the early part of January the patient appeared to have typical jargon aphasia, word-blindness and word-deafness, with gradually increasing bilateral choked disk; all of which began with an apoplectiform onset. The sensory aphasia gradually subsided and has disappeared. Absence of homonymous lateral hemianopia in the presence of the signs of involvement of the left first temporal convolution and angular gyrus, with decided improvement after a comparatively short time, seemed incompatible with the diagnosis of cerebral tumor.

He was treated by repeated lumbar punctures with removal of a small amount of fluid each time, and was given concentrated magnesium sulphate solution on the theory of diminishing intracranial pressure from edema. He seems to have made a complete recovery. I consider this case as probably one of encephalitis of the left temporal lobe.

DISCUSSION

DR. WILLIAM G. SPILLER: The improvement in this man's eyegrounds has been most satisfactory. He permitted only two lumbar punctures, and declined after that to have any further reduction of intracranial pressure in this way. I employed concentrated magnesium sulphate solution by enema in the method suggested by Fay. The magnesium sulphate may be given by mouth or enema. The highest swelling of the disk was 4.5 diopters. Dr. Langdon examined the patient this afternoon and found that the swelling had practically disappeared. I regard this case as one of meningitis serosa or encephalitis.

DR. J. HENDRIE LLOYD: This case appears to be one of vascular obstruction; the sudden apoplectiform onset, the delimitation of the lesion to the language zone, without paralysis, hemianesthesia, or hemianopia; all these features make it look like a vascular lesion. The condition of the eye grounds, as reported, is rather puzzling, but as this has tended to clear up, it evidently does not indicate

a grave organic lesion, whatever the cause may have been. Whether encephalitis can cause such a strictly localized lesion, of such sudden onset, is an interesting question.

DR. WILLIAMS B. CADWALADER: The sudden onset of symptoms did suggest a vascular lesion, but when the choked disk developed I felt that explanation was insufficient. I do not believe the symptoms were due to a vascular lesion alone.

SIMILAR HYSTERICAL CONTRACTURES IN WIFE AND HUSBAND.

DR. T. H. WEISENBURG.

Two cases of hysterical contracture are reported because of their unusual nature and occurrence first in the wife, later being imitated by the husband. The first patient, a Russian Jewess, aged 45, had been in this country 19 years. She had had nine children, two of whom were dead. The patient was well up to six years ago when she noticed a flexor contracture in the fifth digit of the left foot. In a short time, similar flexor contractures of all the toes of this foot developed. Approximately a year later, she noticed that the middle finger of the left hand was flexed, this being soon followed with similar contractures of all the fingers of this hand. These contractures have been present for three years.

The patient complains of constant numbness and needle-like sensation in the affected limbs. The toes are more contracted than the fingers. When her attention is directed elsewhere the contracture is relaxed. It is much easier to relax the contracture in the fingers than in the toes. When the patient is emotional or talks about her troubles, the contractures are worse. This is also true when she is observed. Sensation of all forms is disturbed in the hand and foot and impairment can be easily suggested elsewhere. Neurologic examination shows increase of reflexes and no other symptoms.

The patient is very emotional, dwelling constantly on the ingratitude of her children whom she rarely sees. Investigation by the social service department of the Orthopedic Hospital, where the patients were observed, demonstrated that the patient and her husband were in extreme poverty and were more or less dependent on the charity of the children, which was precarious. The contracture dated from the time when the children left the patient and the poverty began.

The second patient is the husband, who is 47 years of age. He had been a shoemaker and had been earning about \$15 or \$20 a week until three years ago when he lost his trade because of the advent of machinery, and now makes only \$3 or \$4 a week. The contracture is present only in the right hand, having begun about four months ago in the fourth and fifth digits, and is similar to that of his wife. It has gradually extended until it has involved the other fingers, with the exception of the thumb. In the last examination, a beginning flexor contracture was noticed in the second toe of the left foot. The contractures in the husband are not as intense as those in the wife. Sensory disturbances can be found and are altered by suggestion.

It is interesting that the contractures in both the wife and husband began at the time of greatest stress, that is in the woman when the children left her and in the husband when poverty was at its worst. In the woman, the contractures were present in the left hand and foot and of the flexor type, whereas, in the husband, while of the same character, they were in the right hand and are now extending to the left foot. There is no question that they are hysterical in character.

DISCUSSION

DR. J. HENDRIE LLOYD: Cannot this be called an hysterical contracture, an imitative psychosis? I can hardly see how else to explain it. There is a curious condition known as lock spasm, perseveration. I have seen it in a case of gross lesion of the motor region of the brain, in which the patient took a firm grip and could not let loose. But I cannot imagine a husband and wife each having such a lesion in the brain. I should think it was an imitative psychosis.

DR. A. M. ORNSTEEN: I saw this woman a year ago in the service of Dr. Bochiroch at the Samaritan Hospital and she presented the same syndrome that she now shows. We believed at the time that there was a large emotional element in the case. She was always accompanied by her husband who seemed to be very much concerned about her and would watch for the spasm to develop, calling attention to it each time. From the obstetrical position of her hand it was suspected that she might have tetany and I examined the woman's muscles for the spasmophilic electrical reactions, but they were negative. Now it seems that concern and anxiety of the husband about his wife's spasms, and the close association with them has helped him to develop this identical condition purely through suggestion.

JUVENILE GENERAL PARALYSIS WITH ONSET AT SEVEN YEARS.

DR. F. H. LEAVITT.

This patient, a white child aged 8 years, was admitted to the Orthopaedic Hospital and Infirmary for Nervous Diseases, at the request of the mother, who stated that the child's entire manner of life, habits, character and actions had changed and deteriorated since seven years of age. The history of the mental deterioration (in contrast to her former active mental state), the serologic reports, typical of paresis, a family history of syphilis and two miscarriages by the mother, and the physical signs lead to a diagnosis of juvenile general paralysis with its onset at seven years. The family history is that the mother had been treated for syphilis before marriage. Her first child was syphilitic and born dead. The second child was the patient who was born at term after the mother had had antisyphilitic treatment during the entire pregnancy. The third pregnancy resulted in an abortion at three months. The mother's blood reaction was strongly positive, the father's negative.

The patient showed no evidences of syphilis at birth. She began to walk at twelve months of age and to talk at fourteen months. She developed normally into a lively, alert and energetic child, talked well, was obedient and cleanly in her habits and advanced through the second year at school. At seven years of age, her conduct and mentality began to change. She had outbursts of uncontrollable temper, screaming, kicking and endeavoring to destroy everything that came within her reach. She never had any real epileptiform attacks nor paralyses. She has demented steadily since that time, having become practically unmanageable; she pays no attention to the commands of her mother, is restless, and pays no attention to the demands of bowels or bladder and soils her clothes or bed as the case may be. She has forgotten how to dress herself and the uses of knife, fork, and spoon. She eats solely with her hands, spilling much food on herself and the table. She has become cruel and resistive to children and her pets. Her outbursts of violent screaming, fighting and anger occur at any time. She also has spells in which she jumps from bed, dances around,

makes facial grimaces, sings or cries a little, waves her arms in the air then goes back to bed, lies down and sleeps. She had measles at two years, but has had no other infections or accidents.

Physical Examination.—She is a very plump and well developed girl with demented facies. The bridge of the nose is flat. The teeth are widely separated and poorly erupted but not Hutchinsonian in type. She speaks with an indistinct, choreic "thick" voice. There are slow choreo-athetoid movements in the facial muscles and a type of choreiform incoordination in the use of both arms. Her pupils are regular in outline, equal in size, and react well in accommodation, but sluggishly to light. She walks unsteadily, with feet wide apart for a good base of support. The knee jerks are very hyperactive but equal; there are no ankle or patellar clonus and no spastic symptoms. Visceral examination is grossly negative. The Wassermann examination of the blood and spinal fluid showed plus 4 with all antigens; the spinal fluid cell count was 166 and the colloidal gold curve was 5,555,432,210.

GUNSHOT WOUND OF THE LEFT PHRENIC NERVE. DR. F. H. LEAVITT.

This man, a private in the army of Great Britain, aged 27 years, is shown as a case of possible gunshot wound of the left phrenic nerve. The history of the onset of the left chest disability, his symptoms and physical signs, roentgen-ray reports and the anatomic position of the pathway of the bullet are suggestive of left phrenic nerve paralysis. The confusing element is that, as a result of his gun shot wound, he has developed pleural adhesions in the left side of the chest, which interfere with normal respiratory movements; these are shown by fluoroscopic and roentgen-ray studies.

This patient received three perforating bullet wounds during his army service. The first occurred in 1915, in his left forearm. The next, in 1917, perforated his face. The third, in September, 1918, was by a bullet that entered at the left sternoclavicular junction, passed backward and came out at the spine of the scapula at the vertebral border. He was in the hospital for this injury only a comparatively short time. The anatomic position of the wound of entrance was about where the left phrenic nerve enters the mediastinum in close proximity to the subclavian artery and vein.

The patient complains principally of inspiratory dyspnea, especially on muscular exertion. There is marked limitation of movement of the entire left side of the chest and Littel's sign is absent on the left. He experiences difficulty in coughing and sneezing, but suffers no subjective sense of pain or other discomfort.

Roentgen-ray examination, made at the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases revealed: The fluoroscopic examination shows a normal diaphragm on the right side. On the left side, the diaphragm is raised and distorted in its outer third, with two lines running upward and outward that suggest adhesions between the pleura and the diaphragm. This is well shown on inspiration, when the diaphragm is pulled downward. It would seem that the adhesions are not sufficient to cause the marked distortion and collapse, and that some definite paralysis of the diaphragm must also be present. There is slight displacement of the heart to the right. On full expiration, the left side of the diaphragm assumes a more nearly normal shape, while, on full inspiration, the distortion produced by the apparent paralysis is much more marked.

I have never seen a case of gun shot paralysis of the phrenic nerve and this case impressed me as being one of that type; but, because of the confusion incident to the pleural adhesions, the diagnosis is not absolute.

THE SUCCESSFUL REMOVAL OF A SPINAL CORD GROWTH.
DR. TOM A. WILLIAMS.

A woman, aged 39, was referred to me in January, 1923, because of a progressive loss of power in the legs and generally increasing, severe pains, which had begun in September, 1922. Pain had also occurred in the back and there was tingling in the lower part of the abdomen. She had had no acute illness at that time, but in the preceding June, a supposed uterine cancer had been removed by radium without a previous examination. The same surgeon reopened the abdomen in November because of intensity of pain; he had found nothing abnormal in the spine and nothing pathologic was discovered at the operation.

Examination revealed a woman slightly wasted, whose facies portrayed long suffering. She moved the left leg with difficulty but was unable to move the foot. There was marked atrophy of the left calf which measured $12\frac{1}{4}$ inches as compared with $13\frac{3}{4}$ inches on the right. The lower part of the left thigh was also much atrophied, the measurements being $15\frac{1}{2}$ inches at the midway point and 19 inches on the right. The toes could be flexed, but dorsiflexion was practically absent. Both flexion and extension of the knee were much weaker than on the right side, where they were also diminished. The reflexes of the left leg were feeble; on the right, the patellar reflex was much exaggerated and there was ankle clonus, although the toes flexed on stroking the sole. The only disturbance of sensation was some indefinite hyperesthesia in the groins. There were no other neurologic signs. The spinal fluid was normal. The diagnosis was made of an extra medullary neoplasm implicating the third, fourth, and fifth lumbar roots on the left side and compressing the spinal cord at a level as high as the third lumbar segment. In view of the history this was, of course, believed to be a metastasis from the uterus.

Immediate operation was recommended as the best hope of a possible favorable prognosis. The operation which was performed by Dr. Horgan, revealed the dura mater bound down by a mass the size of a large bean at the level of the third lumbar segment. Neither spinal cord nor root was infiltrated and no xanthochromia appeared in the spinal fluid below. The growth was removed and proved to be, not a neoplasm, but a hemorrhagic pachymeningitis of chronic type. In part of the second lumbar vertebra, which appeared soft to the surgeon, no abnormality was found.

Since the operation, no severe pain has been felt by the patient and she has returned home comparatively comfortable. Complete recovery of motility awaits only time for regeneration of the anterior root fibers pressed on by the growth.

TRAUMA OF THE SPINE MISDIAGNOSED AS HYSTERIA. DR. TOM A. WILLIAMS.

The frequency of hysteria as the true interpretation of cases when compensation is in question should not be allowed to blind the physician to other possibilities. Mental inertia permits some men to fall into the error of what I have called diagnosis by categories. Traumatic hysteria was diagnosed by two psychiatrists in the case of a woman who was entitled to treatment by a compensation commission because of an injury to the neck. This was because, after a year's relief, intense pain again ensued with deviation and rigidity of

the neck. The pain and rigidity were not relieved by manipulation, although the patient had been completely relieved previously by manipulative treatment in the same hospital. By these psychiatrists the deviation was looked on as tic, and the whole situation was regarded as a psychic reaction to her former accident. A factor contributing to that diagnosis was the volubility of the patient and her excitable manner.

A more thorough exploration, however, made it clear that the patient's manner reflected, not lack of intellectual balance, but an earnestness of character aroused by a widow's necessity to gain a livelihood for her family. For twenty years she had proved herself highly efficient in responsible clerical work, in which she was eager to continue. Whatever anxiety she showed was attributed to the wish for recovery. All the movements of the head were restricted, more particularly bending to the right. The twist of the neck had none of the characters of mental torticollis. It was merely an attitude adopted to minimize pain.

Tics are convulsive and intemperate in character; are accompanied by consciousness of the act; are preceded by a desire sometimes mounting to a passion to perform the act; and are followed by a feeling of relief after performance of the movement. At all events, the victim of tics feels compelled to make the movement comprising the tic; hence the term *impulsion* or *compulsion neurosis*.

Although the roentgen-ray examination of the neck revealed no displacement of the vertebrae, there could be felt a distinct thickening, on the side of the spine towards which her head was turned, over the left transverse process of the third cervical vertebra. Accordingly I rejected the diagnosis of traumatic neurosis, believing that I was dealing with a trauma of the neck.

The treatment prescribed was manipulation by a skilled operator. The result was that the patient slept that night without the aid of the hypnotics she had been taking. Within a week she was completely free from pain and could move the neck in every direction. She has remained well for six months, although, as a precaution, manipulation was continued weekly.

PARALYSIS OF THE PERONEAL NERVE FROM OVERFLEXION OF THE LEG ON THE THIGH. DR. WILLIAM G. SPILLER.

The peroneal nerve is frequently injured; recently an unusual cause of paralysis of this nerve has come under my observation. A young man was employed in laying hard wood floors and in his enthusiasm for doing the best that he could he over-exerted himself. He knelt on his left knee and kept the right leg flexed on the thigh at more than a right angle, with the sole of foot on the floor, and in this position reached as far forward as possible. In this way he produced severe pressure on the right peroneal nerve by the firmly contracted biceps muscle, the tendon of the muscle being close to the nerve. He developed a complete right sided peroneal palsy. Another man who had been long in the business stated that when he first began the work he also had a paralysis of the peroneal nerve.

CASE OF ACUTE EPIDEMIC ENCEPHALITIS. DR. A. M. ORNSTEEN.

The occurrence of three characteristic symptom-types in rapid succession, with complete recovery in two weeks, are the points of especial interest in this case. The development of a state of acute generalized chorea, severe enough to be regarded as a case of acute chorea gravis, is also noteworthy.

The patient, a previously healthy woman, aged 27, became ill on Feb. 11, 1923, with grippal symptoms, for which she was treated during one week, being

partly confined to bed. On February 18, she suddenly experienced sharp pain in the left shoulder and left abdominal wall, associated with extreme hyperesthesia of the skin over these parts. The pains occurred at frequent intervals, were stabbing in character, and each pain was accompanied by gross jerking of the muscles in these areas. Forty-eight hours after the onset of these myoclonic pains, she suddenly became choreic, irritable and greatly-disturbed emotionally. Within a few hours, every part of the head, body and limbs became involved in coarse, irregular jerking, resembling, in every detail, Sydenham's chorea. Her mental condition was one of great irritability and anxiousness. This continued for forty-eight hours with marked insomnia and signs of beginning physical exhaustion. Great concern was felt about her condition at this time because of the similarity of the condition to the severe cases of acute chorea gravis. The above dyskinetic phenomena stopped as suddenly as they had begun with the appearance of lethargy and diplopia on the beginning of the third day. On the next day, partial bilateral ptosis was noted with slight turning inward of the left eye. The drowsiness and diplopia persisted for three days and then disappeared. Within two weeks from the onset of the severe pains, the patient had recovered completely from a symptomatic point of view. The only persistent symptom during the latter days was hyperesthesia of the left half of the abdomen and the left shoulder.

A rise in temperature was noted only when the chorea subsided and the lethargy appeared; it reached 101 F. by axilla, ran a remittent course for three days, and then only rose in the evening to 99.4 for three more days. A leukocytosis of 13,000 was present. General physical examination of heart and lungs was negative. A spinal fluid examination was not made, the clinical evidence being sufficient for the diagnosis.

ASPHYXIATION AT BIRTH CAUSED BY MENINGEAL HEMORRHAGE. DR. W. B. CADWALADER.

I had a case recently of a child asphyxiated at birth because of difficulty in labor, although forceps had not been used. There were frequent severe general convulsions, as many as thirty in twenty-four hours; lumbar puncture showed bloody spinal fluid. The patient died after three weeks. At post-mortem examination a large subpial blood clot was found covering the anterior surface of the pons and medulla oblongata. My reason for speaking of this case is to emphasize the importance of asphyxiation at birth as a cause of meningeal hemorrhage.

SYPHILIS AS THE CAUSE OF MUSCULAR ATROPHY OF SPINAL ORIGIN. DRs. A. J. OSTHEIMER, GEO. WILSON, AND N. W. WINKELMAN.

After a brief review of the literature in which the writings of Raymond, Léri, Dana and Spiller were especially referred to, the authors reported a number of cases of atrophy of spinal origin in which the evidence clearly pointed toward syphilis as the cause. One of these cases was with necropsy findings. The importance of treating all cases of muscular atrophy of spinal origin with antisyphilitic treatment was stressed. It was the opinion of the authors that this should be done despite negative blood and spinal fluid findings.

* This paper will be published in full at a later date.

DISCUSSION

DR. CHARLES S. POTTS: I wish to add one case to this collection in which the patient was much improved by treatment. I saw this man about 1917. He worked in the Remington Arms Company at Chester where he had to use a hammer continuously. It is interesting that he first noticed weakness in his right arm with which he used the hammer. The atrophy was confined almost entirely to the shoulder girdles and muscles of the back, and there were fibrillary twitchings in these muscles. He had no pyramidal tract symptoms whatever. There was a positive Wassermann reaction; under the use of arsphenamin and mercury the process was arrested and he improved slightly. I saw the man within a year and there had been no progress whatever in his symptoms; if anything he was better.

DR. J. HENDRIE LLOYD: There is no doubt that syphilis can cause muscular atrophy. The wonder is that the spirochete does not invade the anterior horns and anterior roots more frequently than it seems to do. It is a well known fact that muscular atrophy occurs in advanced cases of tabes. I remember a number of such cases in our service at the Philadelphia Hospital; one case, in a woman, I put on record some 20 years ago. Some limbs looked as if she had no muscular tissue left. Nonne, in his monograph on syphilis, has presented pictures of patients with muscular atrophy due to syphilis. There is one phase of this subject to which I want to refer; that is the invasion of the lateral tracts. A few years ago Dr. Ludlum and I recorded cases of what we called primary lateral sclerosis in syphilitic patients. As is well known, lateral sclerosis occurs sometimes in association with muscular atrophy, as in amyotrophic lateral sclerosis. This raises the question whether amyotrophic lateral sclerosis may be due to syphilis.

DR. A. J. OSTHEIMER: I want to stress the importance of the treatment. In view of the inefficacy of therapy of today in all forms of spinal muscular atrophy due to causes other than syphilis, the important point this paper is intended to bring out, is that all muscular atrophies should be treated as if they were due to syphilis. An improvement consequent on such treatment will be also a therapeutic aid in diagnosis. All these cases may be treated in the same way as other neurosyphilis, no matter whether it involves the lateral tracts, the posterior roots and columns, or other portions of the cord or brain.

INDEX TO VOLUME 9

The star (*) preceding the page number indicates an original article in THE ARCHIVES. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B,O and S, respectively.

	PAGE
Aboulker, H.: Diagnosis of silent intracranial abscess in ambulatory stage	648
Abraham, K.: Manifestations of female castration complex.....	521
Actinomycosis, spinal; Lesné and Belloir.....	647
Agersborg, H. P. K.: Qualitative chemical and physical stimulations in nudibranchiate mollusks	377
Agnosia, transitory, of an infant in regard to its arms; Tournay.....	106
Agraphia, left hemiplegia with aphasia in left-handed person, and with agraphia in otherwise unaffected hand; Buckley.....	125
Alajouanine: Septum lucidum tumor.....	524
Alcoholic psychoses before and after prohibition; Pollock.....	378
psychosis associated with brain tumor; Burr.....	120
Amnesia, hysterical amnesia following physical injury; Read.....	533
Amyotonia and myopathy in same patient; Yudelsohn.....	669
Anatomy, experiments on development of cranial ganglia and lateral line sense organs in amblystoma punctatum; Stone.....	104
mutual relationship in development of brain and eyes of Lepidoptera; Kopéc	382
Angioma, venous, of cerebral cortex; Campbell and Ballance.....	235
of pons; Leyser.....	238
Apfelbach, C. W.: Traumatic fractures of cranial bones: edema of brain; bruises of brain	109
Apraxia, localization of; Forster.....	97
Argyll Robertson pupil; Wilson	242
Armstrong, H.: Retrobulbar neuritis with symptoms of spinal sclerosis....	385
Arsphenamin therapy in state hospitals for mental disorders; G. W. Mills and C. L. Vaux.....	*450
Arthritis, neuropathic; Duncan	383
Ascariasis, meningeal symptoms of; Turcan.....	525
Asphyxiation at birth caused by meningeal hemorrhage; Cadwalader.....	829
Asthenia and mania from antityphoid vaccination; Benon.....	800
Atrophy, Charcot-Marie; report of case with necropsy; C. S. Potts and G. Wilson	*431
infantile progressive muscular atrophy of Werdnig-Hoffmann type; case report with necropsy; J. C. Michael.....	*582
muscular, of spinal origin; syphilis as cause of; Ostheimer, Wilson and Winkelman	829
muscular, progressive; Courtney	113
Austria, neurology and psychiatry in; Blitzstein.....	273
Automatism, ambulatory; H. D. Singer.....	*347
Autosuggestion: See Suggestion	
Avelis syndrome; Gordon	121
Ayer, J. B.: Subtentorial cyst of meninges.....	403
Babinski sign, is there a Babinski sign of the hand? Radovici.....	386
Bacillus acidophilus, implantation of, in persons with psychoses; L. A. Julianelle and F. G. Ebaugh.....	*769

INDEX TO VOLUME 9

	PAGE
Bagby, E.: Etiology of phobias.....	523
Bailey, P.: Experimental diabetes insipidus.....	102
Recent developments in electrodiagnosis.....	*436
Unusual tumor of third ventricle in diabetes insipidus.....	807
Baldi, E.: Physiology of nervous system of insects; research on circus movements provoked in coleoptera.....	234
Baldie, A.: Hysteria	383
Ballance, C.: Venous angioma of cerebral cortex.....	235
Barbital intoxication; Stearns, 529; Sands.....	819
treatment of insane epileptics; I. A. Darling.....	*478
Barlow, R. A.: Vestibular nerve function in myxedema.....	525
Bassoe, P., and Davis, C. B.: Two cases of brain tumor with ventricu- lography	*178, 263
Bausch: Elementary convulsions in catatonia.....	95
Belloir: Spinal actinomycosis	647
Benon, R.: Asthenia and mania from antityphoid vaccination.....	800
Berkheiser, E. J.: Neuropathic arthropathies—Charcot's spine.....	385
Bernheim: Pain in gangrene of extremities.....	234
Bertrand: Septum lucidum tumor.....	524
Bessière, R.: Thorium X in psychiatric therapeutics.....	236
Bickel, G.: Recovery from tuberculous meningitis.....	108
Syndrome of the central gray nuclei.....	218
Blastomycosis, cystic, of cerebral gray matter; caused by torula histolytica Stoddard and Cutler; W. Freeman and F. D. Weidman.....	*589
Blitzsten, N. L.: Neurology and psychiatry in Austria and Germany.....	273
Blood creatinin findings in 5 cases of corpus striatum disorder; Raphael and Potter	651
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito.....	792
sugar in blood and spinal fluid; Polonovski and Duhot.....	646
Body and mind: origin of dualism; Mott.....	381
Bolton, G. C.: Hysterical gangrene.....	642
BOOK REVIEWS:	
Anomale Kinder; L. Scholz.....	133
Brain Abscess; Its Surgical Pathology and Operative Technic; W. P. Eagleton	415
Concerning Psychoanalysis; introductory presentation by Allers; edited by Stransky and Dattner.....	673
Constitutional Factors in Dementia Praecox with Particular Attention to the Circulatory System and to Some of the Endocrine Glands; N. D. C. Lewis.....	686
Die Epidemische Encephalitis; F. Stern.....	684
Elements of Scientific Psychology; K. Dunlap.....	132
Functional Nervous Disorders: Their Classification and Treatment; D. E. Core	282
Kingdom of Evils; Psychiatric Social Work Presented in 100 Case His- tories, Together with a Classification of Social Divisions of Evil; E. E. Southard, M. C. Jarrett and R. C. Cabot.....	545
Mental Diseases. A Public Health Problem; J. V. May.....	281
Multiple Sclerosis (Disseminated Sclerosis).....	685
Nervous Ills, Their Cause and Cure; B. Sidis.....	544
Outwitting Our Nerves; J. A. Jackson and H. M. Salisbury.....	416
Psychic Health of Jesus; W. E. Bundy.....	135
Syphilis of the Innocent; H. C. Solomon and M. H. Solomon.....	134
Botulism, epidemic of; Morel and de St. Martin.....	651

INDEX TO VOLUME 9

	PAGE
Boultier: Benzoin colloidal reaction.....	644
Bowman, K. M.: Mental disorder with low basal metabolism.....	259
—and Grabfield, G. P.: Basal metabolism in mental disease.....	*358
Boyd, T. E.: Studies on visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals.....	96
Brain abscess, diagnosis of silent intracranial abscess in ambulatory stage; Aboulker	648
abscess, traumatic, of anterior cerebral fossa; Courtney.....	113
calcification, unusual; Weimann	107
edema, studies in traumatic fractures of cranial bones: edema of brain; bruises of brain; Apfelbach.....	109
fundamental plan of vertebrate brain; Kingsbury.....	108
hemorrhage in young adults; 2 cases; Burley.....	803
mutual relationship in development of brain and eyes of Lepidoptera; Kopéc	382
points of view concerning forebrain morphology in lower vertebrates; Holmgren	111
spirochetes in syphilitic endarteritis of brain; Sioli.....	787
stabilizing brain tissue during fixation; G. S. Stevenson.....	*763
syndrome of central gray nuclei; Bickel.....	218
syphilis and psychoses; Redalié.....	790
tumor, acute alcoholic psychosis associated with brain tumor; Burr.....	120
tumor, cases illustrating some aspects of brain and spinal cord tumors; Elsberg	388
tumor, endothelioma of brain 1 year after operation; Freeman.....	540
tumor, enchondroma of brain removed with recovery; Spiller.....	536
tumor, glioma of cerebral hemispheres; 2 cases; Throckmorton.....	384
tumor, mixed cell sarcoma of; Crafts.....	381
tumor of septum lucidum; Souques, Alajouanine and Bertrand.....	524
tumor, presentation of case in which presumptive diagnosis of tumor had been made 3 years before; Frazier.....	538
tumor, 2 cases with ventriculography; P. Bassoe and C. B. Davis....	*178, 263
tumor, unusual case of brain tumor; Watermann.....	527
tumor, unusual tumor of third ventricle in case of diabetes insipidus; Bailey and Reifenstein.....	807
tumor, venous angioma of cerebral cortex; Campbell and Ballance....	235
Bremer, F.: Experimental diabetes insipidus.....	102
Bresowsky: Lesions of subthalamic region.....	101
Broadwin, J. T.: Spinal spastic paralysis.....	237
Buck, C. F.: Social service work in neurological clinic at the Massachusetts General Hospital during 1922	406
Buckley, A. C.: Left hemiplegia with aphasia in left-handed person, and with agraphia in the otherwise unaffected hand.....	125
Burgess, H.: Basal metabolism in nontoxic goiter and borderline thyroid cases	99
Burley, B. T.: Cerebral hemorrhage in young adults.....	803
Burr, C. W.: Acute alcoholic psychosis associated with brain tumor.....	120
Buscaino, V. M.: Dysthyroidism, anaphylaxis, and epilepsy.....	801
Buzzard, E. F.: Traumatic and toxic ulnar neuritis.....	382
Byrnes, C. M.: Singultus crisis in tabes dorsalis.....	279
Byrnes, M. A.: Study of spinal accessory nerves from a case of bilateral acquired spasmodic torticollis	130
Cadwalader, W. B.: Apoplectiform onset of left temporal lobe symptoms probably due to encephalitis	822
Asphyxiation at birth caused by meningeal hemorrhage.....	829
Herpes zoster and tabes dorsalis.....	119
Paralysis of upward associated movements of eyeballs.....	278
Campbell, H.: Venous angioma of cerebral cortex.....	235
Cannon, W. B.: Experimental traumatic shock; the critical level in falling blood pressure	91

INDEX TO VOLUME 9

	PAGE
Capps, J. A.: Localization of pain sense in parietal and diaphragmatic peritoneum	645
Carbon monoxide (illuminating gas) poisoning: report of case presenting unusual sequela; Wilson	665
Carlson, A. J.: Studies on visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals.....	96
Carniol: Oculogastric and oculocolic reflexes.....	227
Castration complex, manifestations of female castration complex; Abraham	521
Catatonía, elementary convulsions in; Bausch.....	95
schizophrenic, with associated metabolic and vegetative features, report of case; T. Raphael, J. P. Parsons and M. N. Woodwell.....	*471
Catola, G.: Parkinson's disease following encephalitis.....	514
Cattell, M.: Experimental traumatic shock; the critical level in falling blood pressure	91
Central gray nuclei, syndrome of; Bickel.....	218
Cerebellum, atrophy in adults, with report of a clinical case; Arrigo Frigerio	372
demonstration of animals in which results of recent lesions of cerebellar cortex of one side were contrasted with those resulting from extirpation of otic labyrinth of the same side; Pike.....	247
genesis of cerebellar functions; F. Tilney.....	*137, 246
hemorrhage, case of congenital megacolon (Hirschsprung's disease) associated with mental disorder and terminal cerebellar hemorrhage; Lewis	519
tumor: operation: relief of symptoms; Taylor.....	396
Cerebrospinal fluid, benzoin colloidal reaction in 105 neurologic cases; Marie, Bouldier and Iorgulesco.....	644
changes following air injection; Hermann.....	384
reaction of colloidal benzoin in general paralysis; Targowla.....	644
source, distribution and circulation; Stewart.....	243
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito.....	792
sugar in blood and spinal fluid; Polonovski and Duhot.....	646
viscosity of; Soda	650
Cestan: Diagnostic value of spinal fluid Wassermann.....	111
Chadman, M. G.: Myasthenia gravis of slow development.....	116
Charcot joint of elbow in Jewish woman; Lindauer.....	410
Charcot's spine; Ridlon and Berkheiser.....	385
Charcot-Marie atrophy: report of case with necropsy; C. S. Potts and G. Wilson	*431
Charles, J. R.: Manganese poisoning.....	244
Cheney, C. O.: Focal infection: its presence and elimination in functional psychoses	392
Cheney, G. W. H.: Colloidal gold reaction in acute poliomyelitis.....	241
Children, actual environment of children in a Boston district; Taylor.....	260
Chordotomy, section of anterolateral columns of spinal cord (chordotomy); physiologic effects and clinical results in 8 cases; C. H. Frazier and W. G. Spiller	*1
Chorea, clinicopathologic study of acute and chronic chorea; G. Wilson and N. W. Winkelman.....	*170
hemichorea of sudden onset in which there were no other symptoms; Gilpin	664
hemiplegia with hemichorea; Remond and Colombies.....	103
Sydenham's, psychoses associated with; Hammes.....	387
Clark, L. P.: Epileptoid or fainting attacks in hypopituitarism.....	239
Importance of special educational training for mental defect dependent upon organic lesions	373
Clymer, G.: Chronic lead poisoning vs. tabes dorsalis.....	397
Hyoscin in Parkinsonian rigidity.....	399

INDEX TO VOLUME 9

	PAGE
Cole, W. H.: Transplantation of skin in frog tadpoles.....	94
Colella, R.: Association between tuberculosis, neuropsychopathy and delinquency	640
Coleman, G. H.: Localization of pain sense in parietal and diaphragmatic peritoneum	645
Colombies, H.: Hemiplegia with hemichorea.....	103
Colon, congenital megacolon (Hirschsprung's disease) associated with mental disorder and terminal cerebellar hemorrhage; Lewis.....	519
Contracture, similar hysterical contractures in wife and husband; Weisenburg	824
Convulsions, clinical picture and prognosis of frequent minor fits of children; Stier	520
experimental; a consideration of epileptogenous zones of central nervous system; L. J. Pollock.....	*604
studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man; preliminary report; C. A. Elsberg and B. P. Stookey.....	*613
Corpus callosum, results of puncture of; Pohlisch.....	99
Corpus striatum disorder, blood creatinin findings in 5 cases; Raphael and Potter	651
Cotton, H. A.: Functional psychoses.....	223
Courtney, J. W.: Progressive muscular atrophy.....	113
Traumatic abscess of anterior cerebral fossa.....	113
Crafts, L. M.: Mixed cell sarcoma of brain.....	381
Cramer, A.: Recovery from tuberculous meningitis.....	108
Cranium, studies in traumatic fractures of cranial bones: edema of brain; bruises of brain; Apfelbach	109
Cycloplegia in girl of 12; Stearns.....	532
Daday, P.: Thorium X in psychiatric therapeutics.....	230
Da Fano, C.: Changes of Golgi's apparatus in nerve cells of spinal cord following exposure to cold.....	111
Dana, C. L.: Ecology of epilepsy; epilepsy in colonies.....	*551
Danielopolu: Oculogastric and oculocolic reflexes.....	227
Danysz, J.: Origin of psychic energy.....	362
Darling, I. A.: Phenobarbital (luminal) treatment of insane epileptics....	*478
Davenport, C. B.: Ecology of epilepsy; racial and geographic distribution of epilepsy	*554
Davis, C. B., and Bassoe, P.: Two cases of brain tumor with ventriculography	*178, 263
Davis, L. E.: Deep sensibility of face.....	*283
Davis, T. K.: Double papilledema.....	245
Decerebrate rigidity in animals and its recognition in man; Walshe.....	112
on the production of neuro-muscular patterns by release of spinal integrations after decerebration; Kraus and Rabiner.....	239
Delgado, H. F.: Treatment of paresis by inoculation with malaria.....	522
Delinquency, association between tuberculosis, neuropsychopathy and delinquency; Rosolino Colella	640
Dementia praecox, blood analyses in catatonic dementia praecox; Uyematsu and Soda	112
genetic origin of dementia praecox; Mott.....	380
severe changes in nuclei of ganglion cells in dementia praecox; Stocker..	519
sex development and behavior in male patients with dementia praecox; C. E. Gibbs	*73
Depression, case with feelings of unreality; Morris.....	258
Detwiler, S. R.: Studies on retina: photomechanical responses in retina of <i>eremias argus</i>	800

INDEX TO VOLUME 9

	PAGE
Devine, H.: An expiation process in schizophrenia.....	233
Diabetes insipidus, experimental; Bailey and Bremer.....	102
Diaphragm center in cerebral cortex and hiccup; Knapp.....	90
Doyle, J. B.: Glossopharyngeal neuralgia.....	*34
Dualism, origin of; Mott.....	381
Duhot, E.: Sugar in blood and spinal fluid.....	646
Duncan, J. H.: Neuropathic arthritis.....	383
Dunham, H. G.: After-care of infantile paralysis cases of 1916 epidemic in Brooklyn	796
Dunn, A. D.: Pachymeningitis hemorrhagica interna.....	386
Dyspituitarism: See Pituitary	
Dysthyroidism: See under Thyroid	
Dystonia lenticularis; Solomon, 256; Frigerio.....	365
Dystrophy, pathologic findings in heart muscle in progressive muscular dystrophy; J. H. Globus	*59
progressive muscular dystrophy; Friedman.....	236
Ebaugh, F. G., and Julianelle, L. A.: Implantation of bacillus acidophilus in persons with psychoses	*769
Education, importance of special educational training for mental defect dependent upon organic lesions; Clark.....	373
Electrodiagnosis, recent developments in; P. Bailey.....	*436
Elsberg, C. A.: Sensory disturbances in tumors of spinal cord and mem- branes	656
Brain and spinal cord tumors.....	388
—and Stookey, B. P.: Studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man.....	*613
Embolism, occipital lobe embolism; Woods.....	648
Encephalitis, acute epidemic encephalitis; Ornsteen.....	828
apoplectiform onset of left temporal lobe symptoms probably due to encephalitis; Cadwalader	822
clinical considerations on Parkinson's disease following encephalitis; Catola	514
clinical notes on pathology of epidemic encephalitis complicated by a psychosis; G. B. Hassin and D. B. Rotman.....	*22
epidemic, pathologic study of 5 cases including 2 with myoclonia; M. E. Morse	*751
lesions in brain of patient with postencephalitic paralysis agitans; J. C. McKinley	*47
lethargica; Hall	371
pathological findings in chronic progressive encephalitis; Marinesco.....	371
sequels, spasmodic forced respiration as sequel of epidemic encephalitis; Pardee	800
sequels, 4 cases; Ornsteen	667
sequels, review of clinical sequelae of recent epidemic of lethargic encephalitis; Naville	515
status of epidemic encephalitis as an independent disease.....	633
treatment of residual epidemic encephalitis; Hyslop.....	387
twenty cases of lethargic encephalitis with pathological findings in 4 cases; Hume, Natrass and Shaw.....	370
Enchondroma of brain removed with recovery; Spiller.....	536
Endarteritis, spirochetes in syphilitic endarteritis of brain; Sioli.....	787
Endocrines, some endocrine aspects of the psyche (lecture delivered to Upsala Medical Society); Hammar.....	368
Endothelioma of brain 1 year after operation; Freeman.....	540
Enfield, C. D.: Normal sella	387
Epilepsy and bilious attacks; Vining.....	243
and the convulsive state; F. Kennedy.....	*567
ecology of epilepsy; epilepsy in colonies; C. L. Dana.....	*551

INDEX TO VOLUME 9

	PAGE
Epilepsy—Continued	
ecology of epilepsy; racial and geographic distribution of epilepsy; C. B. Davenport.....	*554
experimental convulsions; a consideration of epileptogenous zones of central nervous system; L. J. Pollock.....	*604
focal, 3 cases; Mixter.....	402
mechanism and fundamental cause of epilepsies; J. Rosett.....	*689
pathogenesis of epilepsy; critical review, with new interpretation of available data; M. Osnato.....	254, *488
phenobarbital (luminal treatment of insane epileptics; I. A. Darling..	*478
some problems in; W. Timme.....	*547
studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man; preliminary report; C. A. Elsberg and B. P. Stookey.....	*613
symptomatic epilepsy in birds; an anatomico-pathologic and clinical report; G. Mingazzini	*576
Epileptoid or fainting attacks in hypopituitarism; Clark.....	239
Esophagus, visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals; Carlson, Boyd and Percy.....	96
Eyman, E. V.: Case with unusual mental symptoms.....	662
Face, deep sensibility of; L. E. Davis.....	*283
Fay, T.: Administration of hypertonic salt solutions for relief of intra- cranial pressure	539
—and Grant, F. C.: Lesions of optic chiasm and tracts with relation to adjacent vascular structures	*739
Feeble-mindedness, hereditary and nonhereditary mental defect, a compara- tive study; H. W. Potter and R. S. Viers.....	*339
Flatau, E.: Cervical neurofibroma	524
“Nuquo-mydrasis” sign	523
Forster: Localization of apraxia	97
Foster, N. B.: Metabolic states contributing to uremia.....	*627
Frazier, C. H.: Presentation of case in which presumptive diagnosis of tumor had been made 3 years before.....	538
—and Spiller, W. G.: Section of anterolateral columns of spinal cord (chordotomy)	*1
Freeman, W.: Endothelioma of brain.....	540
Glioma of medulla	541
—and Weidman, F. D.: Cystic blastomycosis of cerebral gray matter.....	*589
Friedman, E. D.: Progressive muscular dystrophy.....	236
Frigerio, A.: Atrophy of cerebellum in adults.....	372
Dystonia lenticularis progressiva	365
Froin's syndrome and its relation to allied conditions in spinal fluid; Greenfield	241
Gangrene, hysterical; Bolton	642
pain in threatened and real gangrene of extremities; Bernheim.....	234
Gaté: Antityphoid vaccination and generalized scleroderma.....	802
Gerard, M. W.: Afferent impulses of trigeminal nerve; intramedullary course of painful, thermal and tactile impulses.....	*306
Germany, impressions of neurology in Russia and Germany; Hassin.....	268
Gibbs, C. E.: Sex development and behavior in male patients with dementia praecox	*73
Gilpin, S. F.: Erb's type of spinal syphilis.....	664
Hemichorea of sudden onset.....	664
Glia reactions, concerning diffuse glia reactions; Pines.....	236
Glioma of medulla; Freeman.....	541
Globus, J. H.: Histopathology of amaurotic family idiocy.....	816
Pathologic findings in heart muscle in progressive muscular dystrophy... Teratoid cyst of hypophysis	*59 *417

INDEX TO VOLUME 9

	PAGE
Glosso-pharyngeal neuralgia, 4 cases; J. B. Doyle.....	*34
Goetsch test of hyperthyroidism.....	635
Goiter, basal metabolism in nontoxic goiter and in borderline thyroid cases, with reference to its bearing in differential diagnosis; Means and Burgess	99
exophthalmic, therapy of.....	781
Goldsmith, W. M.: Process of ingestion in the ciliate, <i>Frontonia</i>	231
Golf, excessive golf followed by musculospiral palsy; Wholey.....	386
Gordon, A.: Avelis syndrome	121
Mother and daughter presenting symptoms of degenerative state of cen- tral nervous system	667
Paramyotonia	276
Sensory disturbance limited to portion of distribution of ulnar nerve.....	411
Grabfield, G. P., and Bowman, K. M.: Basal metabolism in mental disease.*	358
Graff: Pituitrin as muscular tonic.....	107
Grant, F. C.: Peripheral nerve tumor.....	542
—and Fay, T.: Lesions of optic chiasm and tracts with relation to adjacent vascular structures	*739
Grant, S. B.: Tetany	100
Greenfield, J. G.: Froin's syndrome.....	241
Gregg, D.: Physiology of autosuggestion	115
Hall, A. J.: Encephalitis lethargica.....	371
Hallucination, experimental study of mechanism of; Prince.....	232
Hammar, J. A.: Endocrine aspects of the psyche.....	368
Hammes, E. M.: Psychoses with Sydenham's chorea.....	387
Hassin, G. B.: Neurology in Russia and Germany.....	268
Solitary tubercle of spinal cord.....	228
—and Rotman, D. B.: Epidemic encephalitis complicated by a psychosis...*	22
Haviland, C. F.: Mental hygiene.....	369
Hayaschi, Y.: Antiproteolytic ferments, amylase and oxydase in psychosis	637
Hemiplegia, left, with aphasia in left-handed person, and with agraphia in the otherwise unaffected hand; Buckley.....	125
syphilitic, with unusual trophic disturbances; Patten.....	275
with hemichorea; Remond and Colombies.....	103
Henderson, D. K.: War psychoses.....	244
Hermann, G.: Spinal fluid following air injection.....	384
Herpes, association of herpes zoster and tabes dorsalis; Cadwalader.....	119
Hiccup, diaphragm center in cerebral cortex and hiccup; Knapp.....	90
singultus crisis in tabes dorsalis; Byrnes.....	279
Holmes, W. H.: Chronic trophedema (Milroy-Meige disease).....	266
Holmgren, N.: Forebrain morphology in lower vertebrates.....	111
Hospitals, need of psychopathic departments in state hospitals; Ross.....	378
psychiatric institute-hospital; Pilcher	375
Hoyt, R.: Value of neurological examination in syphilis.....	801
Hume, W. E.: Lethargic encephalitis.....	370
Hyperthyroidism: See under Thyroid	
Hypophysis: See Pituitary	
Hyslop, G. H.: Residual epidemic encephalitis.....	387
Hysteria; Baldie	383
hysterical amnesia following physical injury; Read.....	533
hysterical gangrene; Bolton	642
similar hysterical contractures in wife and husband; Weisenburg.....	824
Idiocy, amaurotic family, histopathology of; Globus.....	816

Immunology, studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to the immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito	792
Infection, focal, is the stomach a focus of infection? Kopeloff.....	649
focal; its presence and elimination in functional psychoses; Kopeloff and Cheney	392
Intracranial pressure, administration of hypertonic salt solutions for relief of; Fay	539
Iorgoulesco: Benzoin colloidal reaction.....	644
Jacobi, W.: General protein therapy and active immunization of general paralysis	517
Jacoby, J. R.: Memorial to William M. Leszynsky.....	817
Jahnel, F.: Spirochetes in central nervous system in general paralysis....	511
Jaloustre, L.: Thorium X in psychiatric therapeutics.....	230
Jelliffe, S. E.: Modern school of Dutch neuropsychiatry.....	658
Julianelle, L. A., and Ebaugh, F. G.: Implantation of bacillus acidophilus in persons with psychoses	*769
Keidel, A.: Asymptomatic neurosyphilis; apparent rôle of immunity in genesis of neurosyphilis.....	240
Kennedy, F.: Epilepsy and the convulsive state.....	*567
Extrathecal abscess of spinal cord.....	652
Vertebral dislocation	653
Kingsbury, B. F.: Fundamental plan of vertebrate brain.....	108
Knapp: Diaphragm center in cerebral cortex and hiccup.....	90
Kopéc, S.: Influence of nervous system on development and regeneration of muscles and integument in insects.....	797
Mutual relationship in development of brain and eyes of Lepidoptera....	382
Kopeloff, N.: Focal infection: its presence and elimination in functional psychoses	392
Is stomach a focus of infection?.....	649
Krabbe, K. H.: Tuberosc sclerosis and hydrocephalus.....	105
Kraus, W. M.: Difference between muscular and neuromuscular interpretation of walking	*184
Phylogeny and classification of spinal motor integrations.....	812
Production of neuro-muscular patterns.....	239
Kuhlmann tests; psychiatric importance of; Plant.....	530
Kuntz, A.: Reflex arcs in myenteric and submucous plexuses.....	377
Lead poisoning, chronic, versus tabes dorsalis; Clymer.....	397
Leavitt, F. H.: Gunshot wound of left phrenic nerve.....	826
Juvenile general paralysis with onset at 7 years.....	825
Legrand, R.: Aortitis and trigeminal neuralgia.....	526
Neo-arsphenamin in hypodermic injections.....	229
Lennox, W. G.: Neurosyphilis among Chinese.....	*26
Lesné: Spinal actinomycosis	647
Leszynsky, William M. Leszynsky, memorial to; Jacoby.....	817
Lewis, N. D. C.: Congenital megacolon (Hirschsprung's disease) with mental disorder	519
Leyser: Angioma of the pons.....	238
Morphine poisoning	651
Lindauer, E.: Charcot joint of elbow.....	410
Lipodystrophy, case of; Patten	408
Little's Disease: See Paralysis, spastic	
Locomotor Ataxia: See Tabes	
Lorente y Patrón, S.: Treatment of general paralysis with relapsing fever virus	650

INDEX TO VOLUME 9

	PAGE
McDonald, C. A.: Examination of heart and aortic arch by roentgen rays..	406
McFarland, A. R.: Involvement of nervous system in various phases of syphilis	798
McKinlay, C. A.: Effect of extract of posterior lobe of pituitary on basal metabolism	110
McKinley, J. C.: Lesions in brain of patient with postencephalitic paralysis agitans	*47
Manganese poisoning; 3 cases; Charles.....	244
Mania and asthenia from antityphoid vaccination; Benon.....	800
Marie, P.: Benzoin colloidal reaction.....	644
Marinesco, M. G.: Chronic progressive encephalitis.....	371
Means, J. H.: Basal metabolism in nontoxic goiter and in borderline thyroid cases	99
Medulla, glioma of; report of case with exhibition of specimen; Freeman...	541
Mejía Xesspe, T.: Peruvian folk lore.....	646
Meninges, hemorrhage, asphyxiation at birth caused by; Cadwalader.....	829
meningeal symptoms of ascariasis; Turcan.....	525
subtentorial cyst of meninges, with necropsy; Ayer.....	403
Meningioma, both intradural and extradural, removed from spinal cord with complete recovery; Spiller	534
Meningitis, tuberculous, recovery from; Cramer and Bickel.....	108
Mental defect, hereditary and nonhereditary mental defect, a comparative study; H. W. Potter and R. S. Viers.....	*339
hygiene, actual environment of children in a Boston district; Taylor.....	260
hygiene, facts and fancies of; Pratt.....	808
hygiene needs arising subsequent to school life; Haviland.....	369
symptoms, case with unusual mental symptoms; Eyman.....	662
Metabolism, basal, effect of extract of posterior lobe of pituitary on basal metabolism in normal individuals and in those with endocrine disturbances; McKinlay	110
basal, in mental disease; K. M. Bowman and G. P. Grabfield.....	*358
basal, in nontoxic goiter and in borderline thyroid cases, with particular reference to its bearing in differential diagnosis; Means and Burgess	99
basal, presentation of 5 cases of mental disorder with low metabolism; Bowman	259
metabolic states contributing to uremia; N. B. Foster.....	*627
nature of certain functional nervous disturbances and their treatment along metabolic lines; R. Pemberton.....	*208, 280
study of antiproteolytic ferments, amylase and oxydase in 4 types of psychosis, with especial consideration of metabolism in mental disease; Hayaschi	637
Michael, J. C.: Infantile progressive muscular atrophy of Werdnig-Hoffmann type	*582
Microscopy, stabilizing brain tissue during fixation; G. S. Stevenson.....	*763
Mills, G. W., and Vaux, C. L.: Arsphenamin therapy in state hospitals for mental disorders	*450
Minct, J.: Aortitis and trigeminal neuralgia.....	526
Neo-arsphenamin in hypodermic injection.....	229
Mingazzini, G.: Symptomatic epilepsy in birds.....	*576
Minnich, D. E.: Tarsal sensitivity to solutions of saccharose, in red admiral butterfly, <i>Pyrameis atalanta</i> Linn.....	379
Mixer, W. J.: Focal epilepsy.....	402
Monoplegia, brachial, due to thrombosis of subclavian vein; Wilson.....	333
Moore, J. E.: Asymptomatic neurosyphilis; influence of pregnancy on incidence of neurosyphilis	649
Morel, C.: Epidemic of botulism.....	651
Morphin poisoning, unusual case of; Leyser.....	651
Morris, C. H.: Depression with feelings of unreality.....	258

INDEX TO VOLUME 9

	PAGE
Morse, M. E.: Epidemic encephalitis.....	*751
Motor activity, psychopathologic and localizing value of parallelism between psychic and motor activity; Steck.....	798
Mott, F. W.: Body and mind: origin of dualism.....	381
Genetic origin of dementia praecox.....	380
Movement, difference between muscular and neuromuscular interpretation of walking; W. M. Kraus.....	*184
practical bearing on diagnosis of phylogeny and classification of spinal motor integrations; Kraus	812
Muscles, influence of nervous system on development and regeneration of muscles and integument in insects; Kopec.....	797
Myasthenia gravis of slow development; Spiller and Chadman.....	116
Myoclonias; Roger	797
Myopathy and amyotonia in same patient; Yudelsohn.....	669
Myxedema, vestibular nerve function in; Barlow.....	525
Naito, K.: Spinal fluid and blood of syphilitic and normal persons.....	792
Nathan, P. W.: Lumbar arthritis as cause of sciatica.....	818
Natrass, F. J.: Lethargic encephalitis.....	370
Recurrent hypertrophic neuritis	238
Naville, F.: Sequelae of lethargic encephalitis.....	515
Neo-arsphenamin in hypodermic injections; Minet and Legrand.....	229
Nerve, abducens, researches on abducens nerve and particularly on sig- nificance of its accessory nucleus; Terni.....	783
artificial nerve branches for innervation of paralyzed muscles; Stookey..	249
facial, deep sensibility of the face; L. E. Davis.....	*283
phrenic, gunshot wound of left phrenic nerve; Leavitt.....	826
trigeminal, afferent impulses of trigeminal nerve; intramedullary course of painful, thermal and tactile impulses; M. W. Gerard.....	*306
trigeminal, deep sensibility of the face; L. E. Davis.....	*283
tumor, 2 cases of peripheral nerve tumor; Grant.....	542
ulnar, case of sensory disturbance limited to portion of distribution of ulnar nerve; Gordon	411
Nervous system, influence of, on development and regeneration of muscles and integument in insects; Kopec.....	797
mother and daughter presenting symptoms of a degenerative state of central nervous system; Gordon	667
nature of certain functional nervous disturbances and their treatment along metabolic lines; R. Pemberton.....	*208, 280
physiology of nervous system of insects; research on circus movements provoked in coleoptera; Baldi.....	234
sympathetic, afferent paths of sympathetic system, with special reference to nerve cells of spinal ganglia sending their peripheral processes into the rami communications; Rossi.....	382
syphilis, comparative clinical observations on involvement of nervous system in various phases of syphilis; Stokes and McFarland.....	798
syphilis, Erb's type of spinal syphilis presenting difficulties in diagnosis; Gilpin	664
syphilis, examination of heart and aortic arch by roentgen rays in neurosyphilis; McDonald	406
syphilis, neurorecurrences following treatment with arsphenamin; Zim- mermann	106
syphilis, neurosyphilis among Chinese; W. G. Lennox.....	*26
syphilis, studies in asymptomatic neurosyphilis; apparent influence of pregnancy on incidence of neurosyphilis in women; Moore.....	649
syphilis, studies in asymptomatic neurosyphilis: apparent rôle of immunity in genesis of neurosyphilis; Keidel.....	240
syphilis, value of neurological examination in; Hoyt.....	801
vibrating sensation in diseases of nervous system; Williamson.....	793
visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals; Carlson, Boyd and Pearcy.....	96

INDEX TO VOLUME 9

	PAGE
Neuralgia, glosso-pharyngeal, 4 cases; J. B. Doyle.....	*34
trigeminal, and aortitis; Minet and Legrand.....	526
Neuritis, recurrent, hypertrophic; Nattrass.....	238
retrobulbar, associated with symptoms and signs of disseminated spinal sclerosis; Armstrong	385
ulnar, some varieties of traumatic and toxic ulnar neuritis; Buzzard.....	382
Neurofibroma, cervical; Flatau and Sawicki.....	524
Neurology in Austria and Germany; Blitzsten.....	273
Neuropsychiatry, reflections on the modern school of Dutch neuro- psychiatry; Jelliffe	658
Neuropsychopathy, association between tuberculosis, neuropsychopathy and delinquency; Rosolino Colella	640
Neurosphilis: See Nervous System, syphilis	
Nicholas: Antityphoid vaccination and generalized scleroderma.....	802
Nixon, C. E.: Spinal fluid and blood of syphilitic and normal persons.....	792
"Nuquo-mydrasis" sign; Flatau	523
Nystagmus, significance of nystagmus in a lesion of abducens nucleus; Rosett	232
 OBITUARIES:	
Edward Mercur William, M.D., 1881-1823.....	778
Robert Thaxter Edes	506
Occupational therapy as means of controlling disturbed patients; Ryon....	385
Optic chiasm and tracts, lesions of, with relation to adjacent vascular structures; T. Fay and F. C. Grant.....	*739
Ornstein, A. M.: Acute epidemic encephalitis.....	828
Postencephalitic sequelae.....	667
Osnato, M.: Pathogenesis of epilepsy.....	254, *488
Ostheimer, A. J.: Syphilis as cause of muscular atrophy of spinal origin...	829
 Pachymeningitis hemorrhagica interna: study of 5 cases of nontraumatic hemorrhagic spinal fluid; Dunn.....	386
Pain, localization of pain sense in parietal and diaphragmatic peritoneum; Capps and Coleman	645
Palsy: See Paralysis	
Papilledema, double, caused by blocking of cord at fourth cervical vertebra, greatly relieved by operation; Davis.....	245
Paralysis agitans in girl of 12; Stearns.....	531
agitans, clinical considerations on Parkinson's disease following enceph- alitis; Catola	514
agitans, hyoscin in Parkinsonian rigidity; Clymer.....	399
agitans, lesions in brain of patient with postencephalitic paralysis agitans; J. C. McKinley	*47
artificial nerve branches for innervation of paralyzed muscles; Stookey.....	249
Avelis syndrome; Gordon	121
Brown-Sequard paralysis from dislocation of cervical vertebra; Viets....	399
excessive golf followed by musculospiral palsy; Wholey.....	386
familial infantile bulbar palsy; Paulian.....	526
general protein therapy and active immunization of general paralysis; Jacobi	517
general, reaction of colloidal benzoin in general paralysis; Targowla....	644
general, spirochetes in central nervous system in general paralysis; Jahnel	511
general, treatment with relapsing fever virus; Lorente y Patrón.....	650
general, treatment by inoculation with malaria; Wagner-Jauregg, 522; Delgado	522
juvenile general paralysis with onset at 7 years; Leavitt.....	825
of peroneal nerve from overflexion of leg on thigh; Spiller.....	828
of upward associated movements of eyeballs; Cadwalader.....	278
spastic, some ocular phases of Little's disease; Posey.....	802
spinal spastic paralysis (primary lateral sclerosis); J. T. Broadwin.....	237

INDEX TO VOLUME 9

	PAGE
Paramyotonia, case of; Gordon.....	276
Pardee, I. H.: Spasmodic forced respiration as sequel of encephalitis....	800
Paresis: See Paralysis, general	
Parker, G. H.: Crawling of young loggerhead turtles toward sea.....	243
Parkinson's disease: See Paralysis agitans	
Parsons, J. P., Woodwell, M. N., and Raphael, T.: Schizophrenic catatonia with associated metabolic and vegetative features.....	*471
Patten, C. A.: Lipodystrophy	408
Syphilitic hemiplegia	275
Paulian, E. D.: Familial infantile bulbar palsy.....	526
Pearcy, J. F.: Visceral sensory nervous system; reflex control of cardia and lower esophagus in mammals.....	96
Pellagra, two cases; Smith.....	257
Pemberton, R.: Functional nervous disturbances and their treatment along metabolic lines	*208, 280
Peruvian folk lore; Mejía Xesspe.....	646
Pfahler: Calcification of pituitary with hypopituitarism and with symp- tomatic treatment	240
Pfister, O.: Plato: a forerunner of psychoanalysis.....	525
Phobia, etiology of; Bagby	523
Physiology, crawling of young loggerhead turtles toward the sea; Parker... process of ingestion in the ciliate, frontonia; Goldsmith.....	243 231
Pick's visions; Stenvers	794
Pike, F. H.: Animals in which the results of recent lesions of cerebellar cortex of one side were contrasted with those resulting from extirpa- tion of the otic labyrinth of same side.....	247
Pilcher, L. F.: Psychiatric institute-hospital.....	375
Pines, J. L.: Diffuse glia reactions.....	236
Pitfield: Calcification of pituitary with hypopituitarism and with symp- tomatic treatment	240
Pituitary, calcification of pituitary with hypopituitarism and with symp- tomatic treatment; Pfahler and Pitfield.....	240
epileptoid or fainting attacks in hypopituitarism; Clark.....	239
extract, effect of extract of posterior lobe of pituitary on basal metabolism in normal individuals and in those with endocrine disturbances; McKinlay	110
extract, further facts regarding influence of feeding anterior lobe of hypophysis on rate of growth and size of ambystoma tigrinum; Uhlenhuth	802
teratoid cyst of hypophysis; J. H. Globus.....	*417
Pituitrin as muscular tonic; an ergographic study; Urechia and Graff.....	107
Plant, J. S.: Psychiatric importance of Kuhlmann tests.....	530
Pohlisch: Results of puncture of corpus callosum.....	99
Polio myelitis, after-care of infantile paralysis cases of 1916 epidemic in Brooklyn; Dunham	796
value of routine use of colloidal gold reaction in acute epidemic polio myelitis; Regan and Cheney.....	241
Pollock, H. M.: Alcoholic psychoses before and after prohibition.....	378
Pollock, L. J.: Experimental convulsions; consideration of the epilep- togenous zones of central nervous system.....	*604
Polonovski: Sugar in blood and spinal fluid.....	646
Pons, angioma of; Leyser	238
tubercle of tegmentum pontis—contribution to symptomatology of pontine lesions—Pick's visions; Stenvers	794
Popper, E.: Cause of schizophrenia.....	104
Development of schizophrenia	93
Posey, W. C.: Ocular phases of Little's disease.....	802

INDEX TO VOLUME 9

	PAGE
Potter, F. C.: Blood creatinin findings in corpus striatum disorder.....	651
Potter, H. W., and Viers, R. S.: Hereditary and nonhereditary mental defect	*339
Potts, C. S., and Wilson, G.: Charcot-Marie atrophy.....	*431
Pratt, G. V.: Facts and fancies of mental hygiene.....	808
Prince, M.: Experimental study of mechanism of hallucinations.....	232
Psychoanalysis. Plato: a forerunner of psychoanalysis; Pfister.....	525
Psychiatry, histopathologic studies in; Spielmeier.....	639
Psychic activity, psychopathologic and localizing value of parallelism between psychic and motor activity; Steck.....	798
energy, origin of; Danysz.....	362
Psychology in medicine; Wells.....	376
Psychopathic inferiority, constitutional; Visser.....	375
Psychosis, alcoholic, before and after prohibition; Pollock.....	378
alcoholic, associated with brain tumor; Burr.....	120
and cerebral syphilis; Redalić.....	790
associated with Sydenham's chorea; Hammes.....	387
basal metabolism in mental disease; K. M. Bowman and G. P. Grabfield.....	*358
clinical and pathologic differentiation of senile psychoses and arterio-sclerotic psychoses; Vonderahe	122
etiology and treatment of the so-called functional psychoses; summary of results based upon experience of 4 years; Cotton.....	223
implantation of bacillus acidophilus in persons with psychoses; L. A. Julianelle and F. G. Ebaugh.....	*769
preliminary study of the precipitating situation in 200 cases of mental disease; Strecker	102
presentation of 5 cases of mental disorder with low basal metabolism; Bowman	259
studies in focal infection; its presence and elimination in functional psychoses; Kopeloff and Cheney.....	392
study of antiproteolytic ferments, amylase and oxydase in 4 types of psychosis, with special consideration of metabolism in mental disease; Hayaschi	637
war; the infective exhaustive group; Henderson.....	244
Puberty, precocious, tuberosc sclerosis and hydrocephalus and their association with precocious puberty; Krabbe.....	105
Pulawski, A.: Thymic death.....	799
Pupil, Argyll Robertson; Wilson	242
Pyramidal tracts, injury of one pyramidal tract resulting from knitting needle puncture wound of cervical region of cord; Yaskin.....	413
Rabiner, A. M.: Production of neuro-muscular patterns.....	239
Radovici, A.: Is there a Babinski sign of hand?.....	386
Oculogastric and oculocolic reflexes.....	227
Palm-chin reflex	98
Raphael, T.: Blood creatinin findings in corpus striatum disorder.....	651
—Parsons, J. P., and Woodwell, M. N.: Schizophrenic catatonia with associated metabolic and vegetative features.....	*471
Raynor, M. W.: Social work in state hospital.....	242
Read, C. F.: Hysterical amnesia following physical injury.....	533
Ream, M. J.: Temperament in harmonious human relationship.....	524
Redalić, L.: Cerebral syphilis and psychoses.....	790
Reflex arcs, occurrence of, in myenteric and submucous plexuses; Kuntz....	377
oculogastric and oculocolic reflexes; Danielopolu, Radovici and Carniol..	227
palm-chin reflex; Radovici	98
Regan: Colloidal gold reaction in acute poliomyelitis.....	241
Reifenstein, B. J.: Unusual tumor of third ventricle in diabetes insipidus...	807
Reimond, A.: Hemiplegia with hemichorea.....	103

INDEX TO VOLUME 9

PAGE

Retina, studies of retina; photomechanical responses in retina of eremias argus; Detwiler	800
Ridlon, J.: Neuropathic arthropathies—Charcot's spine.....	385
Riser: Diagnostic value of spinal fluid Wassermann.....	111
Roger, H.: The myoclonias.....	797
Rosett, J.: Mechanism and fundamental cause of epilepsies.....	*689
Significance of nystagmus in a lesion of abducens nucleus.....	232
Ross, J. R.: Need of psychopathic department in state hospitals.....	378
Rossi, O.: Afferent paths of sympathetic nervous system.....	382
Rotman, D. B., and Hassin, G. B.: Pathology in case of epidemic encephalitis complicated by a psychosis.....	*22
Russia, impressions of neurology in; Hassin.....	268
Ryon, W. G.: Occupational therapy to control disturbed patients.....	385
Saccharose, quantitative study of tarsal sensitivity of solutions of saccharose, in red admiral butterfly, <i>Pyrameis atalanta</i> Linn; Minnich	379
de St. Martin, R.: Epidemic of botulism.....	651
Sands, I. J.: Barbitol intoxication.....	819
Sawicki, B.: Cervical neurofibroma	524
Schizophrenia, and expiation process in case of schizophrenia; Devine....	233
clinical studies on cause of; Popper.....	104
clinical studies on development of; Popper.....	93
schizophrenic catatonia with associated metabolic and vegetative features; T. Raphael, J. P. Parsons and M. N. Woodwell.....	*471
Sciatica, lumbar arthritis as cause of; Nathan.....	818
Scleroderma, antityphoid vaccination and generalized scleroderma; Nicholas and Gaté	802
Sclerosis, combined, of spinal cord of anemic type without blood changes; Winkelman	412
multiple, spirochete stain in multiple sclerosis; G. S. Stevenson.....	*88
Sella turcica, normal sella; Enfield.....	387
Sergeant's cutaneous white line; Sézary.....	110
Sézary: Cutaneous white line (of Sergeant).....	110
Shaw, A. F. B.: Lethargic encephalitis.....	370
Shock, experimental traumatic shock; the critical level in falling blood pressure; Cannon and Cattell.....	91
Singer, H. D.: Ambulatory automatism.....	*347
Anosmia and agensia.....	262
Sioli, F.: Spirochetes in syphilitic endarteritis of brain.....	787
Skin, transplantation of skin in frog tadpoles, with reference to adjustment of grafts over the eyes, and to local specificity of integument; Cole... ..	94
Skull: See Cranium	
Smell, an unusual syndrome; anosmia and agensia; Singer.....	262
Smith, S. K.: Pellagra	257
Social service, organization of social work in state hospital; Raynor.....	242
resumé of social service work in neurological clinic at Massachusetts General Hospital during 1922; Buck.....	406
SOCIETY TRANSACTIONS:	
Boston Society of Psychiatry and Neurology.....	113, 256, 396, 527, 803
Chicago Neurological Society	262, 531, 669
New York Neurological Society.....	245, 388, 652, 812, 817
New York Neurological Society and Neurological Section of the Academy of Medicine	248
Philadelphia Neurological Society.....	116, 275, 408, 534, 662, 822
Soda, T.: Blood analyses in catatonic dementia praecox.....	112
Viscosity of cerebrospinal fluid.....	650

INDEX TO VOLUME 9

	PAGE
Solomon, H. C.: Dystonia lenticularis.....	256
Souques: Septum lucidum tumor	524
Spilmeyer, W.: Histopathologic studies in psychiatry.....	639
Spiller, W. G.: Enchondroma of brain removed with recovery.....	536
Meningioma, both intradural and extradural, removed from spinal cord..	534
Myasthenia gravis.....	116
Paralysis of peroneal nerve from overflexion of leg on thigh.....	828
—and Frazier, C. H.: Section of anterolateral columns of spinal cord (chordotomy)	*1
Spinal cord, abscess, case of extrathecal abscess of cord; Taylor and Kennedy	652
changes of Golgi's apparatus in nerve cells of spinal cord following exposure to cold; Da Fano.....	111
section of anterolateral columns of spinal cord (chordotomy); discussion of physiologic effects and clinical results in series of 8 cases; C. H. Frazier and W. G. Spiller.....	*1
sensory disturbances in tumors of spinal cord and membranes, and their diagnostic significance; Elsberg	656
tubercle, clinico pathological notes on solitary tubercle of spinal cord; Thalheimer and Hassin.....	228
tumor, meningioma both intradural and extradural, removed from spinal cord with complete recovery; Spiller.....	534
tumor, successful removal of spinal cord growth; Williams.....	827
tumors, cases illustrating some aspects of brain and spinal cord tumors; Elsberg	388
Spine, case of vertebral dislocation; reduction; Taylor and Kennedy.....	653
trauma of spine misdiagnosed as hysteria; Williams.....	827
Spirochetes in syphilitic endarteritis of brain; Sioli.....	787
Stearns, A. W.: Barbitol intoxication	529
Sterns, W. G.: Cycloplegia in girl of 12.....	532
Paralysis agitans in girl of 12.....	531
Steck, H.: Psychopathologic and localizing value of parallelism between psychic and motor activity	798
Stenvers, H. W.: Tubercle of tegmentum pontis.....	794
Stevenson, G. S.: Spirochete stain in multiple sclerosis.....	*88
Stabilizing brain tissue during fixation.....	*763
Stevenson, L.: Comparative study of icebox and water bath fixation methods in Wassefmann as applied to nervous diseases.....	248
Stewart, R. M.: Spinal fluid; source, distribution, and circulation.....	243
Stier, E.: Prognosis of frequent minor fits of children.....	520
Stocker, A.: Severe changes in nuclei of ganglion cells in dementia praecox	519
Stoddart, W. H. B.: Mental factor in visceroptosis.....	387
Stokes, J. H.: Involvement of nervous system in various phases of syphilis.	798
Stomach, is the stomach a focus of infection? Kopeloff.....	649
Stone, L. S.: Experiments on development of cranial ganglia and lateral line sense organs in amblystoma punctatum.....	104
Stookey, B.: Artificial nerve branches for innervation of paralyzed muscles	249
—and Elsberg, C. A.: Studies on epilepsy; convulsions experimentally produced in animals compared with convulsive states in man.....	*613
Strecker, E. A.: The precipitating situation in 200 cases of mental disease..	102
Suggestion, physiology of autosuggestion; Gregg.....	115
Syphilis: See also Nervous system, syphilis of	
arsphenamin therapy in state hospitals for mental disorders; G. W. Mills and C. L. Vaux.....	*450
cerebral, and psychoses; Redalié	790
studies of cerebrospinal fluid and blood of syphilitic and normal persons with special reference to immunity reactions and colloidal gold test on original and unfiltered fluids and serums; Nixon and Naito....	792

INDEX TO VOLUME 9

	PAGE
Tabes dorsalis, association of herpes zoster and tabes dorsalis; Cadwalader	119
chronic lead poisoning versus tabes dorsalis; Clymer	397
singultus crisis in tabes dorsalis; Byrnes	279
Targowla, R.: Reaction of colloidal benzoin in general paralysis	644
Taste, an unusual syndrome: anosmia and agensia; Singer	262
Taylor, A. S.: Extrathecal abscess of spinal cord	652
Vertebral dislocation; reduction	653
Taylor, E. W.: Cerebellar tumor: operation: relief of symptoms	396
Taylor, M.: Actual environment of children in a Boston district	260
Temperament in harmonious human relationship; Ream	524
Teratoid cyst of the hypophysis; J. H. Globus	*417
Termopsis, castes of; Thompson	109
Terni, T.: Researches on the abducens nerve	783
Tetany; report of cases with acid-base disturbance; Grant	100
Thalamus, contribution to our knowledge of lesions of subthalamic region; Bresowsky	101
Thalhimer, W.: Solitary tubercle of spinal cord	228
Thompson, C. B.: Castes of Termopsis	109
Thorium X in psychiatric therapeutics; Daday, Bessière and Jaloustre	230
Throckmorton, T. B.: Glioma of cerebral hemispheres	384
Thymus, new contribution to study of thymic death; Pulawski	799
Thyroid, dysthyroidism, anaphylaxis and epilepsy; Buscaino	801
hyperthyroidism, test of	635
Tilney, F.: Genesis of cerebellar functions	*137, 246
Timme, W.: Epilepsy	*547
Torticollis, study of spinal accessory nerves from case of bilateral acquired spasmodic torticollis; Byrnes	130
Tournay, A.: Transitory agnosia of infant	106
Trophedema, chronic trophedema (Milroy-Meige disease); report of 2 cases; Holmes	266
Tuberculosis, association between tuberculosis, neuropsychopathy and delin- quency; Rosolino Colella	640
tubercle of tegmentum pontis—contribution to symptomatology of pontine lesions—Pick's visions; Stenvers	794
Tumor: See Brain tumor; Pons, tumor; Spinal cord tumor, etc.	
Turcan, H.: Meningeal symptoms of ascariasis	525
Typhoid vaccination and generalized scleroderma; Nicholas and Gaté	802
vaccination, asthenia and mania from; Benon	800
Uhlenhuth, E.: Influence of feeding anterior lobe of hypophysis on rate of growth and size of ambystoma tigrinum	802
Urechia: Pituitrin as muscular tonic	107
Uremia, metabolic states contributing to; N. B. Foster	*627
Uyematsu, S.: Blood analyses in catatonic dementia praecox	112
Vaux, C. L., and Mills, G. W.: Arsphenamin therapy in state hospitals for mental disorders	*450
Ventriculography, spinal fluid changes following air injection; Hermann	384
two cases of brain tumor with; P. Bassoe and C. B. Davis	*178, 263
Vertigo and its treatment; Woolsey	400
Vibrating sensation in diseases of nervous system; Williamson	793
Viers, R. S., and Potter, H. W.: Hereditary and nonhereditary mental defect	*336
Viets, H. R.: Brown-Sequard paralysis from dislocation of cervical vertebra	399
Vining, C. W.: Bilious attacks and epilepsy	243



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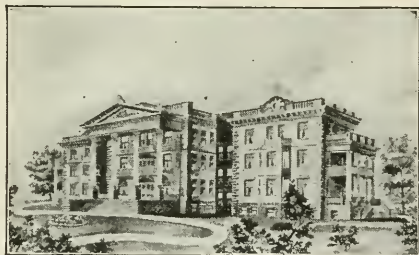
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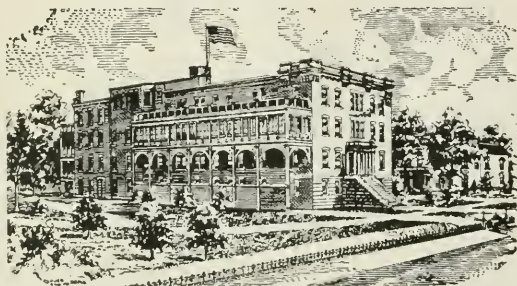
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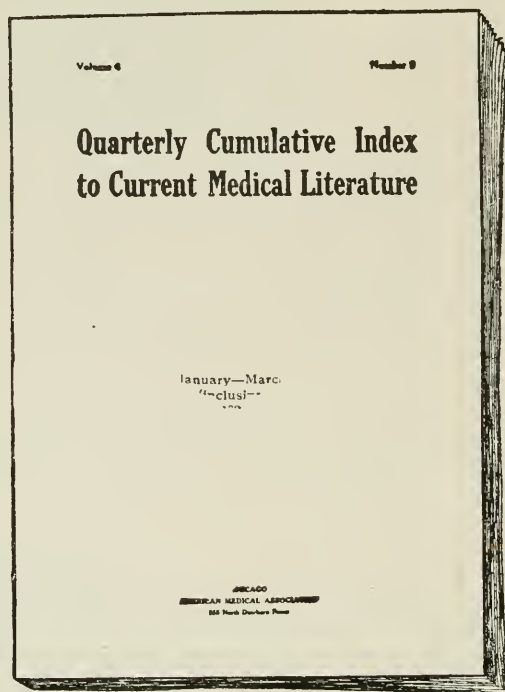
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